

This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

#### Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + Refrain from automated querying Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

#### **About Google Book Search**

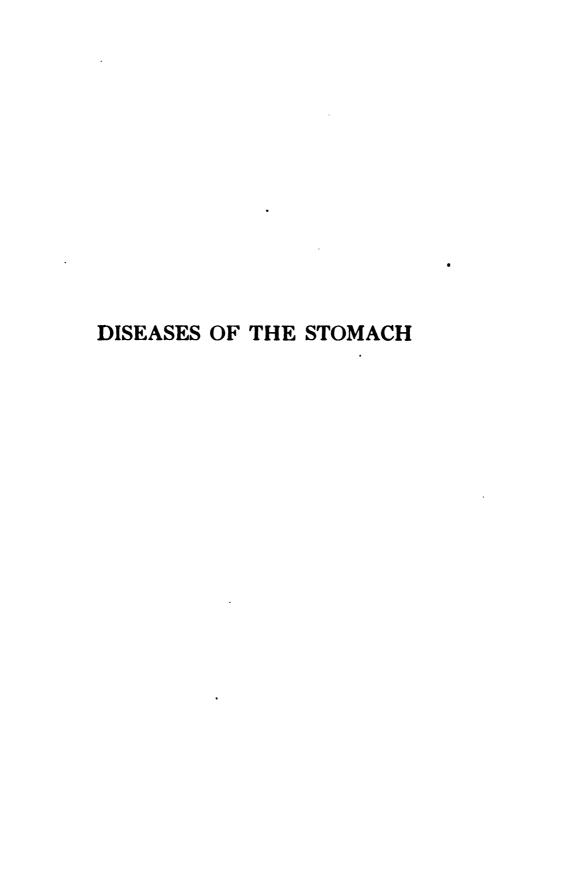
Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at http://books.google.com/



# BOSTON MEDICAL LIBRARY 8 THE FENWAY









# DISEASES OF THE STOMACH

# AND THEIR RELATION TO OTHER DISEASES

BY  $\smile$ 

#### CHARLES G. STOCKTON, M.D.

PROFESSOR OF MEDICINE, MEDICAL DEPARTMENT, UNIVERSITY
OF BUFFALO; ATTENDING PHYSICIAN, BUFFALO
GENERAL HOSPITAL, ETC.

WITH FIVE PLATES, TWENTY-TWO RADIOGRAMS AND SIXTY-FIVE ILLUSTRATIONS IN THE TEXT



D. APPLETON AND COMPANY NEW YORK AND LONDON 1914

### COPYRIGHT, 1914, BY D. APPLETON AND COMPANY



Printed in the United States of America

# TO THE MEMORY OF DR. CHARLES LEWIS STOCKTON MY FATHER AND FIRST TEACHER IN MEDICINE



#### PREFACE

In writing this book no attempt has been made to avoid the personal note; informality of style may be excused because this is largely a record of personal experience. However, conclusions are based not only on experience in my clinic, but on my acquaintance with the clinics of colleagues whose work is deservedly valued. When it has served a special purpose, I have referred to the publications of others, sometimes liberally, but usually without intent to summarize prevailing professional opinion.

The book is written primarily for the use of general practitioners, not forgetting that numerous body one time my pupils, but consideration also is given to the needs of the beginners.

I am indebted to Dr. Allen A. Jones for the use of his material, for reading the manuscript and for very helpful suggestions; to Dr. H. U. Williams for a concise review of the pathology of gastric cancer and for assistance in other ways; to Dr. Kavaleff Mankell for a selective and condensed description of physical methods of treatment; to Dr. Karl F. Eschelman for a description of laboratory tests; to Dr. Gaylord, of the State Institute for Study of Malignant Disease, for access to statistics; to Drs. Jacobs, Simpson, Russell, Schreiner and others for preparation of material used in illustrations. To Dr. Leonard Reu belongs the credit for most of the radiograms employed, and I have to thank Dr. Ward Plummer for several important plates.

I am grateful to numerous friends, foreign and

American, for the privilege of reproducing illustrations. Finally, I owe much to Dr. Rose Donk for invaluable assistance in preparing the book for press.

CHARLES G. STOCKTON.

Buffalo, N. Y.

#### INTRODUCTION

Some explanation may be demanded of one who ventures to put forth a book on diseases of the stomach at a time when there are numerous recent treatises on the subject, and when the profession is intent upon the surgical treatment of important gastric diseases. Here then is the "raison d'être" for this book.

When one has for twenty-five years taught and practiced general medicine it would be remarkable if he had not accumulated notions, derived in study and observation from a personal viewpoint, which might be read with interest by other workers. When in addition to this, during the same time, special attention had been given to the subject of abdominal diseases, it would be disappointing if there had not grown out of this association something of practical importance to the physician, surgeon, and pupil and perhaps to the special worker. At any rate these chapters are addressed to each of these; and, for the reason that, in the writer's judgment, diseases of the stomach represent a field where the various sides of the profession have mutual interest, there would seem to be occasion for a discussion of the subject written from the standpoint of the internist, rather than from that of the pure specialist.

Diseases of the stomach, although more or less closely bound with most departments of medicine, occupy the domain of the general practitioner and the internist. These clinicians justly hold that the problems involved belong primarily to their own broad field. I have held that when the internists come to devote to gastric pa-

thology the close study and the broad view which are requisite for a productive understanding of the subject, there will cease to be a demand for the stomach specialist. That time, however, has not arrived. The contributions to our knowledge made by the specialist are of immense importance; their skill in diagnosis and treatment is at present indispensable. The general practitioner and the surgeon seem not to appreciate the importance of familiarity with this subject. This expert knowledge, so often slighted, holds for them invaluable assistance in general diagnosis. Unquestionably the specialist would be the better had he a wide experience in general medicine; undoubtedly the diagnostician in internal medicine is not adequately equipped unless he has made himself proficient in diseases of the stomach.

As for the surgeon, called upon to relieve men from the peril of gross structural disease, sometimes to make the best of the situation left by the physician, it is but natural for him to exhibit some impatience with methods that may seem to be dilatory if not even uncalled for. A few, fortunately only a few, take the extreme position that the stomach is of little practical importance to the economy. They regard it as an organ prone to disease, yet serving little physiologic need, save as a reservoir, like the crop of a fowl. Of course, when pressed, they admit that it is more than a crop, that it serves, also, as a gizzard; perhaps something even besides this; yet they discuss the possibilities of surgery of the stomach as if the function of that organ were a negligible quantity.

The future would probably accord more credit to the medicine of our era if we held more tenaciously to the guidance of nature.

The stomach is prone to serious organic disease. Is

this not indicative that it is an organ of supreme functional activity? A part but moderately endowed with functional activity, escapes, proportionately from disease.¹ An organ appointed to perform special functions is provided with specialized structure for their expression. The functions of the stomach are varied, and numerically greater than we formerly supposed; it is highly specialized; embryologically speaking, some of the structures are comparatively recent. The problems of gastric pathology are not merely unsuccessfully managed, some of them are yet unrecognized. Let us, therefore, exercise modesty when we deal with the unknown, and so far the unsurmountable.

Among the more conspicuous nihilistic announcements that may be heard is this, that gastric digestion is unimportant and unnecessary. This assertion comes from research workers, internists and surgeons alike.

It has been pointed out that with absence both of acid and enzyme secretion by the stomach that digestion is carried on by other enzymes, in the alkaline intestinal medium, without previous aid from the stomach, and that under these conditions, nutrition seems to be unimpaired. It is held that, when the stomach is removed from animals the organism soon adapts itself to the loss, and digestion by the intestines seems to be adequate. Chittenden, in his studies in nutrition, found that a few apparently healthy and vigorous individuals had ordinarily, low gastric secretion, and exceptionally one would be found with no gastric secretion whatever.

Were we to accept this view that gastric digestion is

<sup>&</sup>lt;sup>1</sup> A critic remarks that the appendix vermiformis, having but little functional activity, is also prone to disease. I would reply that, aside from infection, the appendix is largely exempt from structural changes and, so far as known, it escapes from functional disturbances.

needless it would follow that nature has added a superfluous function, or else that, as a safeguard, like the tailor who adds a second seam she has reinforced the resources of digestion, so that the necessary digestion might not be endangered.

The matter can hardly be disposed of thus summarily, and however much truth there may be in the conception of an auxiliary digestion, or an emergency and vicarious digestion, there are grounds for disbelieving in the use-lessness or the unimportance of gastric proteolysis. It may be assumed with reasonable safety that, when nature invariably resorts to a definite physiological method, when she develops an apparatus for this special functional activity, she has an essential and indispensable need of that function.

This applies to gastric digestion or, at least, to mineral acid and enzyme proteolysis.

It has been shown that this type of digestion belongs not alone to the higher animals, but that it is carried on in an analogous method by the unicellular organisms. The matter is perhaps best stated in the language of Prof. Gary N. Calkins of Columbia (Science, Aug. 4, 1911).

"The early observations by Le Dantec, Meissner, Fabre-Domergue, Greenwood and others from 1888-1894 demonstrated the presence of some mineral acid in connection with proteid digestion in different types of protozoa, and it was suggested that some simple ferment, acting in an acid medium, is responsible for digestion in these single cells. This suggestion was confirmed by Hartog and Dixon in 1901, who isolated a proteolytic ferment active in an acid medium; but the subject became more complicated when Mouton and Mesnil in 1902-03 isolated a proteolytic ferment that was active in an alkaline medium, and suggested that the digestive ferment in

protozoa is more like trypsin than pepsin. Finally, Nierenstein and Metalnikoff, in 1903-07, showed that both types of ferment are involved, digestion beginning with an acid reaction, followed by an alkaline reaction, and conforming in a general way with the digestive processes in higher animals. Few physiologists have attacked the problem of assimilation in protozoa. Verworm, however, in his 'Biogenhypothese,' has outlined a theoretical conception of the combination of protoplasm molecules with the products of proteid digestion, based on the Erhlich side-chain hypothesis.''

The fact that cellular activity includes a definite digestive function through the intervention of enzymes, some of which act by means of an alkaline medium and others by means of an acid medium, would indicate that the process is based on fundamental necessities. would further appear that in the development of the stomach we witness the introduction of a specialized apparatus adopted in the interest of economy of energy and as a means of utilizing a wider range of proteid foods which, otherwise, might be noxious. It would appear that the cellular metabolism of proteids bears somewhat the relation to the stomach that carbohydrate metabolism in the cells bears to the function of the intestine, liver and pancreas. It is highly probable that the reason that animals and men may exist after excision of the stomach is owing to the fact that cellular activity is competent, when under the necessity, of carrying on a sufficient part of this work, thus assisting in the maintenance of the balance of nutrition.

Should this prove to be so it would follow, nevertheless, that an animal without a stomach, or a man without gastric digestion would be handicapped. (See Achylia Gastrica.) That is, he would become more dependent

upon general "cellular digestion," and in case of any general strain, as in fever or unusual physical or mental stress of any sort, he would fail; and that much more quickly than the individual possessed of a helpful stomach.

Besides this it is probable that he would be unusually exposed to the damaging results of the introduction of proteids unmodified by the acid proteolysis of the stomach.

Now in point of fact, that is precisely what I have observed to be the case in victims of the condition which we call achylia gastrica. After years of study of this subject in a relatively large number of cases, I cannot say that I ever encountered one who could be classed as a healthy person. Having no common group of symptoms, making no complaint that would indicate a definite disease,—they seem, nevertheless, to lack stamina, to be unequal to long sustained efforts, to be especially liable to perturbations of nutrition, and, I have thought, since that question has been brought to our notice, that they are especially liable to what is called food anaphylaxis or allergy.

That recent acquisition, our beginning knowledge of the internal secretions or gastric hormones, although as yet scarcely more than recognized, serves to warn that the facts of digestion as related to the stomach cannot be apprehended by short measure consideration.

Late to attract attention, these hormones were probably biologically the earliest agents through the action of which the stomach was enabled to exercise the several functions of secretion and motion.

It is only recently that we have appreciated the full significance of motility. In realizing this, the tendency comes to minimize the importance of secretion. We

should be reluctant to depreciate the need of any part of a function with which we are equipped. While it may be proper to eliminate a certain factor in a working hypothesis, for the time being, the whole truth of a question is likely to await the result of study that embraces the multiple working hypothesis. The more I study the question of digestion in health and disease, the more I am impressed with its implications and complications; the more necessary it seems for physicians to acquire a comprehensive and minute knowledge of the subject. It is not easy to prescribe in this a course of study for a student; he must live with and work at the subject throughout his career. At least there is wisdom in indicating to him that a proper conception of pathology demands an ever present consideration of nutrition and elimination. It would be well for him to be imbued with the belief that these are bound up with the question of digestion; then he may understand more readily that the organism is strung with relations that connect the stomach with many organs, most apparatuses and all the systems.

The intimate association between diseases of the stomach and other diseases, between gastric symptoms and disturbances of remote parts, has been given further attention in the section on dyspepsia. In fact, a belief that this relationship deserves further emphasis than it has received represents the chief reason for the production of this book.

The incidence of disease of the stomach is closely wrapped up with the subject of general experience, the plane of vital activity in which the individual moves. There is place for a work on sociology, economics and nutrition. The social and personal life of an individual being intimately known, the history of his digestive apparatus may be largely forecast. The alimentation of a

people has both a direct and indirect effect upon gastric digestion. In nations where economic conditions greatly limit, almost eliminate, the ingestion of animal proteids, diseases of the stomach are less frequently encountered. This is not alone because of the diet, yet the character of the food is an element in the result. In America where the laboring people are accustomed to eat meat daily, digestive disturbances are more frequent than in the peasantry of Germany or France, where bread and vegetables compose the chief aliment. The cost of meat has already risen to a point that has decreased its consumption; ultimately this factor will greatly diminish the eating of meat, and this doubtless will lead to improvement in public health.

The belief that the eating of coarse food is per se detrimental, is a misapprehension; it may be either harmful or beneficial; all depends upon other intercurrent factors. The statement is made that the stomach of middle life complains because of the indigestible food that was taken cheerfully in youth. This also is a misapprehension. The very fact that certain foods are slowly digested and assimilated renders them preferable to individuals of energy and great physical activity and such foods are well adapted to those who have power of rapid oxidation.

A lumberman, exposed to a northern winter, his strong frame at full activity, prefers his meal of boiled pork, "flap-jacks," molasses and dried apple pie; it proves to be sufficient and lasting; he does not feel gnawing hunger before the next meal hour. Such a meal engenders no dyspepsia in him; but to a seamstress, stitching for long hours in a confined, over heated chamber, it would be disastrous. In her case, as in that of the pampered, lazy and luxurious, it is necessary, if symptoms are to be

avoided, to have the requisite number of food units of such character, and so prepared, that hydration and assimilation become easy.

A common error is the belief that restricting the diet to delicate foods is to benefit the healthy stomach, in that it is spared effort. As well contend that a man is better without exercise. A stomach may lose in efficiency, become more and more intolerant, as less is required of it; and, conversely, its energy and tolerance may be built up by a consistent increase in the quality and quantity of food taken at regular repasts, with due respect for restful abstinence between meals.

In acting upon these suggestions the important matter of caring for the proper number of food-units from day to day must be remembered. The questions of denutrition and repletion are not always readily disposed of. Much depends upon the even and harmonious distribution and discharge of energy; and this is a matter that involves the inheritance, the environment and the habits of the individual. The exigencies of conventionality, livelihood, school and business requirements here raise themselves. The question of wage and disbursement, of play and labor, of happiness and responsibility, each has an emphatic bearing upon digestive processes. working of a superstitious mind, of an over-sensitive organization and of a physical handicap has to be considered. Sometimes dyspepsia is rather the result than the cause of bad humor, suspicion, jealousy, insomnia and discontent. The affairs of life are ordered for the convenience of a school, a shop or an office, not for the best interest of pupil or employes. A wise old man once remarked to me that "tasks are classified crosswise, rather than lengthwise." That is true. The work required is estimated on the basis of the endurance of the average, not on that of the vigorous and precocious, or of the frail and under developed.

Observe a crowd of children at the close of the afternoon hour, or a street full of operatives when the five o'clock whistle blows, and note the difference in individual demeanor. You may venture judgment on the state of digestion in each. The exuberant, the contented, the lagging, the sluggard, the suffering, each is fitted for a particular, not for a common job. Yet often the same results are demanded of each, and the same portion of the same variety of food is, in some instances, provided for each. This is notably true in armies, public institutions of all kinds, in boarding houses, and, strange to say, in many families. Absence of a sense of proportion enters into the causes of diseases of the stomach; a statement that applies to the psychic as much as to the physical state.

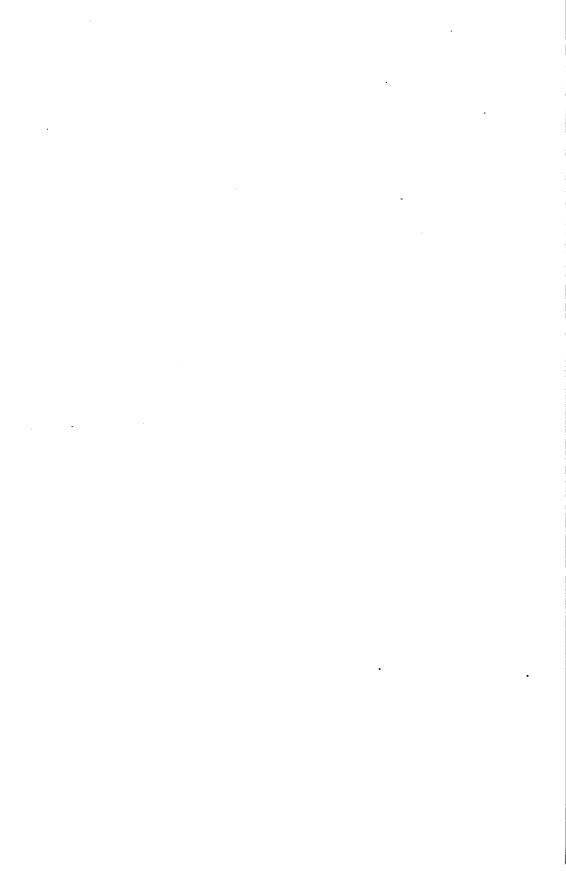
It may be remarked that indigestion often begins in the head. This comprehensive view of the nature of functional disorders of the stomach is, of course, an old story to experienced physicians, who perceive that the statements apply to functional diseases in general, to exhaustion, neurasthenia, etc. However, the student is less aware of this, and too often we older men overlook it.

There is an old contention that what we have been accustomed to regard as functional disease, has in fact an unrecognized structure basis. I am of the opinion that many functional troubles are the expression of anatomical changes, no matter how minute; on the other hand, there can be no question but that physiologic stress and disharmony, such as we name functional disease, is a potent cause of morbid structural alteration.

There was a time when morbid anatomy was considered all important in pathology; when the well trained

mind inquired with Virchow: "Where is the disease?" Great as was this advance it does not mark the destination of pathologic research. We recognize that structural changes are rather the result of disease, not the disease. Just as anatomical defects conflict with normal physiologic activity, so physiologic weakness or strain induces structural degeneration.

In regard to diseases of the stomach it is profitable to remember this. Not always can we draw the delimitation of disease. Just now we are encouraged to illuminate the obscure province of nutrition by studies in organic chemistry, metabolism and immunity, the physiology of the nervous system and psychology; and doubtless we shall obtain a clearer view of the mechanism of nutrition and further exemplification of the mystery of vitality.



#### **CONTENTS**

														PAGE
Introduction	•	•	•	•		•						•	•	ix
		CF	TAI	PT	ER.	т								
4 37 4 00							• ^ •							
ANAT	OM	ΙΥ	AN	D.	РН	YS.	101	<b>700</b>	łΥ					
Stomach	•	•	•	•	•	•	•	•	•	•	•	•	•	3 3
Structure	•	•	•	:		•	•	•	•	•	•	•,	•	6
Lymphatics	•		•		:		•	•	:	•	•	•	•	10
The Blood Supply	:			:			:	:	:	:	•	:	•	10
The Nerve Supply														11
GASTRIC SECRETION .														19
		n <b>u</b>	AT	TE	'D	TT								
T37 4 3 (1)							** 4							
EXAMI	NA	TIC	JN	OF	T	1E	PA	TI	EN'	Г				
PERSONAL HISTORY .	•		•	•	•	•	•	•	•	•	•	•	•	24
PHYSICAL EXAMINATION		•	•	•	•	•	•	•	•	•	•	•	٠	30
		•		•	•	•	•	•	•	•	•	•	٠	31
Palpation Percussion	•	•	•		•	•	•	•	•	•	•	•	٠	33 43
Auscultation	•	•	•	•	•	•	•	•	•	•	•	•	•	45
ruscultation	•	•	•	•	•	•	•	•	•	•	•	•	•	ΨU
	C	H.	$\mathbf{AP}$	TE	$\mathbf{R}$	Ш								
THI	E G	AS	TR	IC	СО	NT	EN	TS						
GASTRIC JUICE								•						47
TEST MEALS														48
											•			48
Chemistry of the C	last	ric	Co	onte	nts									<b>4</b> 9
Carcinoma		•		•	•		•		<b>'</b> •	•	•	•		61
Microscopical Exam				•				•	•	•	•	•		62
TESTS FOR MOTOR ACTIVI	TY (	OF ?	THE	ST	OM	ACH	•	•	•	•	•	•	•	63
	(	CH.	ΑP	TE	R	ΙV								
Tr.	не	SI	ron	MA(	СН	т	BE	2						
CLINICAL VALUE TECHNIQUE OF USING T								•						67 73
·				PTE										
		_					1 PR							
THE STRING TEST	ıН	E	STF	RIN	G '	LES	ST							77
THE BIRING LEST	•	•	•	٠.	•	•	•	•	•	•	•	•	•	"
			2	xxi										

#### CONTENTS

#### CHAPTER VI

DUODENAL ALIMENTATIO	NC				PAGE
DUODENAL ALIMENTATION	•		•, •	•	80
CHAPTER VII					
RADIOGRAPHY AND FLUORO	SCOP	V			
Introduction		_			83
DOUBLE CAPSULE MEANS OF RECOGNIZING SLOW	Morro	ONT .	• •	•	89
THE INTERPRETATIONS OF RADIOGRAMS				•	90
Normal Stomach	•	•	•	•	90
Normal Stomach	•			•	93
Pylorie Spasm	•			•	94
Obstruction of the Pylorus	•	•		•	94
Adhesions from Pericholecystitis				•	94
Over-tonic Stomach				•	95
Over-tonic Stomach					95
Atonic Stomach				•	95
Gastrectasis				•	. 96
Gastrectasis					96
Duodenal Ulcer				•	98
Duodenal Ulcer					100
				Ĭ	
CHAPTER VIII					
DYSPEPSIA					
Introduction					101
VAGOTONIC AND SYMPATHETICOTONIC STATES					104
FOOD POISON, AUTO-INTOXICATION, ANAPHYLAXI	8.				108
Metabolism and the Liver					111
Metabolism and the Liver					113
Treatment					115
Treatment					116
Diagnosis of Cecal Stasis					118
The Course of a Mild Attack of Cecal Stagn	ation				118
Treatment					119
Treatment					120
GASTRIC DISTURBANCES SECONDARY TO CHRONIC	APP	ENDI	CITIS		122
THE HEART AND DYSPEPSIA					
DYSPEPSIA FROM IRRITATION OF THE URINARY T	BACT				128
NEURASTHENIA AND DYSPEPSIA					129
NEURASTHENIA AND DYSPEPSIA					131
Treatment					133
INTERNAL SECRETIONS AS RELATED TO DYSPEPSI	Α.				133
DIATHESIS AND DYSPEPSIA					135
DIATHESIS AND DYSPEPSIA					138
DISTURBANCES OF THE SPECIAL SENSES IN RELATI	ON TO	Dy	SPEP	SIA	139
Eye-strain and Dyspepsia					140

CONTENTS	xxiii
	PAGE
GASTRITIS AND FUNCTIONAL DYSPEPSIA	. 145
THE NATURE OF DYSPEPSIA	. 145
TREATMENT OF DYSPEPSIA	. 146
CHAPTER IX	
FUNCTIONAL DERANGEMENTS OF DIGESTION AND TRIC NEUROSES	GAS-
Introduction	. 154
SYMPTOMS AND SIGNS	. 155
VAGOTONIC AND SYMPATHETICOTONIC STATES	. 155
Types of Functional Disturbances	. 156
Anomalies of Secretion	. 156
Motor Disturbances	. 170
Motor Disturbances	. 185
$\mathbf{CHAPTER} \ \mathbf{X}$	
THE STOMACH IN RELATION TO OTHER DISEAS	ES
	. 199
	. 200
STOMACH IN TUBERCULOSIS	203
STOMACH IN TUBERCUROUS	210
RENAL DISPASES IN RELATION TO THE STONAGE	919
THE STOWAGE IN TERMINATE TO THE STOWAGE	914
THE STOMACH, IN FERRILE CONDITIONS	217
THE STOMACH IN RELATION TO ADTUDITION	218
RELATION RETWEEN SKIN DISEASES AND DIGESTION	220
THE STOMACH IN DIABETES	223
	. 220
CHAPTER XI	
PEPTIC ULCER	
	. 228
Sex	. 229
OCCUPATION AND GEOGRAPHICAL DISTRIBUTION	. 229
MORBID ANATOMY	. 229
Recent Peptic Ulcer	
Chronic Peptic Ulcer	. 233
The Erosions	938
PATHOLOGY AND PATHOGENESIS	. 200 946
Summary	240
CHEMICAL COURSE AND SYMPTOMATOLOGY	. 270
Ulcer With Abrupt Onset	210
Recurring Ulcer	. 286
Chronic Ulcer	. 287
Drodonal Illeer	901
Duodenal Ulcer	205
Differential Diagnosis	308 . 209
Differential Diagnosis	. 500

.

#### CONTENTS

## CHAPTER XII PEPTIC ULCER

	Con	tinı	ued								1	PAGE
TREATMENT												311
TREATMENT	it of	F	Іур	era	cidi	ty,	Ну	per	seci	retio	n	319
and Irregular Secreti Indications for Treatment	of	Μo	tor	Ė	ceita	bili	ty,	Gas	stri	· U	n-	012
rest, Spasm, Obstruct Indications for the Treatn												
stance and Hemorrh Indications for Treatment	age											327
and the Contiguous P	ptic	U	cer									343
COMPLICATIONS AND SEQUELAI	OF	G.	ASTE	RIC	$\mathbf{U}_{\mathbf{L}}$	CER						347
Reichmann's Symptom-Co	omol	ex	and	l it	ts I	Rela	tion	ı ta	G	astr	ic	
Ulcer							•		•			347
Perforating Ulcer												350
Ulcer Perforating Ulcer Pyloric Stenosis and Obst	ruct	ion	•	•	•	•	•	•	•	•	•	360
CHA	<b>\PT</b>	ΈF	R X	II	I							
GAST												
Introduction	•		•	•	•	•	•	•	•	•		372
SYMPTOMATOLOGY	•	•	•	•	•	•	•	•	•	•	•	377
TREATMENT	•	•	•	•	•	•	•	•	•	•	٠	377
CH	API	ΈI	R A	ZIX	7							
CANCER OF THE STOMA						L C	ON	SII	Œ	RAI	ì.	ons
AND												
INTRODUCTION												378
INCIDENCE OF CANCER OF THE	ST	OM.	ACH									381
The Localization of Cance	er as	to	the	O	rga	ns A	\ffe	cted	١.			383
Pathology •	•											397
Anatomy	•		•									400
Histology and Classifica	tion	•	•		•	•		•		•		402
Metastases	•	•	•	•	•	•	•	•	•	•	•	404
Metastases CLINICAL HISTORY AND SYMUNUSUAL FORMS OF CANCER (	PTOM OF T	IAT HE	olo Sto	JY MA	С <b>н</b>	•	:	:	:	:	•	404 422
СН	AP'	ТE	R	ΧV	7							
CANCER OF THE STOMA	CH:	D	IAG	N(	osi	S A	NI	Т	RE	AT	M)	ENT
DIAGNOSIS OF CANCER												
Special Diagnosis of Can	.cer	•	•	:	•	•	•	•	•	•	•	441
Prognosis							•					
TREATMENT	•		•	•	•	•		•	•	•		453

CONTENTS													
CHAPTER XVI													
TUMORS OF THE STOMACH OTHER THAN CARCINOMAT													
SARCOMA OF THE STOMACH	PAGE . 460												
	. 460												
Clinical History	. 462												
Diagnosis	. 462												
Treatment	. 463												
	. 463												
Adenomata and Polypi	. 464												
False Tumors	. 465												
roreign bodies	. 466												
	. 467 . 468												
ACTINOMICOSIS OF THE STOMACH	. 400												
CHAPTER XVII													
HEMATEMESIS AND GASTRORRHAGIA													
HEMATEMESIS FROM BLEEDING OUTSIDE THE STOMACH													
HEMATEMESIS FROM BLEEDING WITHIN THE STOMACH. GAS													
	. 471												
	. 474												
The Symptoms of Gastrorrhagia	. 475												
reatment	. 410												
CHAPTER XVIII													
GASTROSUCCORRHEA													
GASTROSUCCORRHEA CONTINUA. PERSISTENT SECRETION OF GAS													
TRIC JUICE (Riegel)	. 481												
Symptoms	. 482												
Prognosis													
Tiognoss	. 483												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) .	. 484												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) . DIGESTIVE GASTROSUCCORRHEA	. 484 . 485												
Gastrosuccorrhea Periodica. Gastrocynsis (Rossbach) .  Digestive Gastrosuccorrhea	. 484 . 485 . 486												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach)  DIGESTIVE GASTROSUCCORRHEA	<ul><li>484</li><li>485</li><li>486</li><li>487</li></ul>												
Gastrosuccorrhea Periodica. Gastrocynsis (Rossbach) .  Digestive Gastrosuccorrhea	<ul><li>484</li><li>485</li><li>486</li><li>487</li></ul>												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach)  DIGESTIVE GASTROSUCCORRHEA	<ul><li>484</li><li>485</li><li>486</li><li>487</li></ul>												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA	. 484 . 485 . 486 . 487 . 487												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA	. 484 . 485 . 486 . 487 . 487												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA	. 484 . 485 . 486 . 487 . 488 . 490												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA	. 484 . 485 . 486 . 487 . 488 . 490 . 492												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA	. 484 . 485 . 486 . 487 . 487 . 488 . 490 . 492 . 493												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA Diagnosis TREATMENT OF GASTROSUCCORRHEA THE EFFECT OF EYE-STRAIN  CHAPTER XIX  MOTOR INSUFFICIENCY—GASTRIC ATONY INTRODUCTION HORMONES RELATION BETWEEN ATONY AND DILATATION TEMPORARY GASTRIC ATONY SYMPTOMATOLOGY AND CLINICAL COURSE	. 484 . 485 . 486 . 487 . 487 . 488 . 490 . 492 . 493												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA Diagnosis TREATMENT OF GASTROSUCCORRHEA THE EFFECT OF EYE-STRAIN  CHAPTER XIX  MOTOR INSUFFICIENCY—GASTRIC ATONY INTRODUCTION HORMONES RELATION BETWEEN ATONY AND DILATATION TEMPORARY GASTRIC ATONY SYMPTOMATOLOGY AND CLINICAL COURSE DIAGNOSIS	. 484 . 485 . 486 . 487 . 487 . 488 . 490 . 492 . 493 . 493												
GASTROSUCCORRHEA PERIODICA. GASTROCYNSIS (Rossbach) DIGESTIVE GASTROSUCCORRHEA Diagnosis TREATMENT OF GASTROSUCCORRHEA THE EFFECT OF EYE-STRAIN  CHAPTER XIX  MOTOR INSUFFICIENCY—GASTRIC ATONY INTRODUCTION HORMONES RELATION BETWEEN ATONY AND DILATATION TEMPORARY GASTRIC ATONY SYMPTOMATOLOGY AND CLINICAL COURSE DIAGNOSIS PROGNOSIS	. 484 . 485 . 486 . 487 . 487 . 488 . 490 . 492 . 493 . 493												

#### CONTENTS

#### CHAPTER XX

GASTRECTASIS. DILATATION OF THE STOMACH	
	PAGE
Introduction	<b>500</b>
· · · · · · · · · · · · · · · · · · ·	DOT
DILATATION FROM ATONY	201 201
DILATATION FROM STENOSIS	DOT
ETIOLOGY	502
Anatomical Changes	504
CLINICAL COURSE AND SYMPTOMS	504
DIAGNOSIS AND OBJECTIVE SYMPTOMS	507
Prognosis	512
Treatment	513
CHAPTER XXI	
ACUTE GASTRECTASIS	
	<b>515</b>
Introduction	51Q
IREATMENT	013
CHAPTER XXII	
AEROPHAGY AND GASEOUS DISTENSION	
	ະດດ
Introduction	022 E04
Treatment	<b>524</b>
. CHAPTER XXIII	
GASTRIC TETANY	
	EOR
	<i>84</i> 0
Diagnosis	ຍ <u>⊿</u> 0 ຮາດ
Prognosis	UAU EGG
TREATMENT	92 <del>9</del>
CHAPTER XXIV	
	177
GASTROPTOSIS—ENTEROPTOSIS—ABDOMINAL MASSAC	
Introduction	530
Gastroptosis	537
Symptoms and Diagnosis	<b>540</b>
Gastroptosis	<b>542</b>
CHAPTER XXV	
ABDOMINAL MASSAGE	
ABDOMINAL MASSAGE	<b>551</b>
	_

				CC	)N	TE.	NT	S							x	xvii
			CH	AI	<b>PT</b> :	ER	X	χv	71							
				G.	AS'	TR)	TI	S								PAGE
GENERAL CONSIDE	ERAT	TON	g					_								
Classification				•	•	•	•	:		•	•		•	•	•	555 557
ACUTE CATARRHA	л. G	اعدا	י ייים דיו	170	•	•	•	•	•	•	•	•	•	•	•	557
Etiology .		I AU	11011	10	•	•	•	•	•		•	•	•	•	•	559 560
Morbid Anat	· ·	•	•	•	•	•	•	•	•	•	•	•	•			
Clinical Cou	и Ма	an ć		·	· nto		•			•	•	•	•			561
Diagnosia	100	аш		уш	ρω	шв	•	•		•	•	•	•			562
Diagnosis . Treatment	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	565
Toxic Gastritis	•	•	•	•	•	•	•	•	•	•	•	•	•			566
ACUTE PHLEGMON	•			•		•	•	•	•	•	•	•	•	•	•	568
THOLOGE I HIEGMON	1008	U/	ASTE	HTI	B	•	•	•	•	•	•	•	•			568
Etiology .	•	•	•	•	•	•	•	•	•		•	•	•	•	•	569
Morbid Anat	omy	3	CI	•		•	•	•	•	-	•	•	•	•	•	569
Clinical Cour	rse a	na	Sy.	mpi	tom	8	•	•	•		•	•	•	•	•	570
Prognosis .	•	•	•	•	•	•	•	•	•	•	•	•	•			571
Treatment	•	٠,	•	•	•	•	•	•	•		•	•	•	•	•	571
CHBONIC CATARRI	HAL	G.	STR	ITI	3	•	•	•	•		•	•			•	<b>572</b>
Morbid Anat	omy		•	•	•	•	•	•	•	•	•	•	•	•	•	572
Etiology .	•	•	•	•			•	•	•		•	•				577
Clinical Cour	se a	nd	Syı	npi	tom	8	•	•		•	•			•	•	579
Diagnosis and	d Di	ffer	rent	ial	Dia	agn	osis	•	•	•						585
Prognosis .	•		•	•		•	•		•							588
Treatment						•				-	•					<b>590</b>
ALCOHOLIC GASTI			•						•		•					600
Symptoms	•															601
Treatment	•	•									•					602
		_	<b>~</b> TT		· -		<b></b> -									
		(	CH.	ΑP	TI	ΣK	$\mathbf{X}^{\mathbf{X}}$	X V	11							
		A	CH	YL	ΙA	G.	AS7	[R]	[CA							
NATURE AND HIS	TORY	•							_							604
ETIOLOGY			•	•	•	•	•	•	•	•	•	•	•	•	•	608
TREATMENT .		•	•	:			•	•	•	•	•	•	•	•	•	612
	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	014
		C	H	\P	TE	$\mathbf{R}$	XX	V	II							
ARTERIOSCLE	יסמי									770	700	171 T	α -	· 17.7	PF -	TT.
THEORIGINA	Unu	OT	5 11	N V	TO TO	M V	NUT CHET	11	ıĽ	٧J	ממה	بلظ	<b>5</b> (	JĽ'	TI	1E
T																
Introduction .	•	•					•	-	•	•	•	•	•			615
DIAGNOSIS		•														618
TREATMENT .	•	•	•	•	•	•	•	•		•	٠	•	•	•	•	618
			СН	ΑF	<b>T</b>	ER	X	ΧI	X							
	SYF									[A	сн					
Introduction .					_											enn
			•	•		•	•	•	•	•	•	•	•	•	•	620
TREATMENT .										-						625 627
	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	U26

#### xxviii

#### CONTENTS

#### CHAPTER XXX

	Ρ.				STE												PAGE
Introduction																	628
Symptoms .				•													630 630
TREATMENT					•		•		•	•			•				<b>630</b>
				CF	IAI	<b>PT</b> :	ER	$\mathbf{X}$	XX	I							
					v	OΜ	(IT	INC	1								
T.,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,					-				-								con
Introduction Cerebral Vol	•	•	•	•	•	•	•	•		•				•		•	632 633
VOMITING FRO	MIT	U.a	·	. T	DDIE	n a <i>m</i>	•	•	•			•		•			634
UREMIC VOMI														:	•	•	634
REFLEX AND	Upa	urdi.	·	vr	Vo:	· WTT	ING	•	•	•	•	•	:			•	635
Vomiting fro	Mr.	Eve	-91	. V & 1D A T1	NT O	W111	IN G	•	•	•	•	•	•	•	•		636
Sympathetic	Vo	MIT	TNC	ieni,		•	•	•	•	•	•	•	•	•	•	•	636
Vomiting from	w I	Иет	ARO	T.IC	ĊA	USE	:g	•	•	•	•	•	•	•	•	•	636
Post-traumat	TC .	AND	Po	)8T-	OPEI	RAT	IVE	Ϋ́ο	М I Т	'ING	•	•	•	•			637
TREATMENT																•	637
VOMITING OF	Pre	MN.	ANC	Y	•	•	•	•	•			•	•	•	•	•	639
CYCLIC OR RE	CUR	RIN	ι I	7ом	TTIN	ıG					•	•		•	•		643
Cyclic Vo	mit	ing	of	Ch	ildh	ood								•	•	•	644
Cyclic Vo	mit	ing										•		•	•	•	646
Treatment	t																647
				OT:	T	·m 1	<b>.</b>	323	LF 37								
				-	AF												
A	BI	001	IIN	IAI	P	AIN	I A	ND	Tl	ENI	Œ	RNI	ess	}			
Source And	Сн	ARA	CTI	ER													649
VARIOUS PATH	10L	OGIC	AL	Co	NDI	TIO	NS		•		•		•		<b>.</b>		653
				~~		m		***									
			•	_	AP					_							
				TI	ΗE	ES	OP	HA	GU	JS							
STRUCTURE AN OBSTRUCTION	1D	Fu	NCT	ION													666
OBSTRUCTION	OF	TH	е Е	Csor	PHA	GUS											668
Cardiospa	smı	us															670
Obstructio	on l	Due	to	St	ruct	ure	ıl D	)ise	ases	з.							681
CANCER OF TH Diagnosis	ie I	Esoi	PHA	GUS	3.						•						690
Diagnosis																	691
Treatment RUPTURE OF T	t																692
RUPTURE OF T	HE	Esc	OPH	ΑGι	S	•											693
Traumatic Spontane	e R	upt	ure									•			•		<b>6</b> 93
Spontane	ous	Ru	ptu	ıre	of t	he	Eso	pha	ıgu	š.			•			•	693
Symptom	ato!	logy	7			•			•		•				•		696
Prognosis								•		•		•	•	•	•		697
Treatmen	t		•		•				•	•	•	•	•	•	•		697
Symptom Prognosis Treatment DIVERTICULA	)F I	HE	$\mathbf{E}\mathbf{s}$	OPE	LAGU	JS	•	•		•	•	•	•	•	•	•	697

				CC	)N	re:	NT	S							XXIX PAGE
Diagnosis .												_			. 697
Treatment				·		•									. 697
ESOPHAGITIS .															. 698
Acute Esop															. 698
Chronic Esc														•	. 700
CORROSIVE DESTR															. 702
PARALYSIS AND															. 702
<b>Treat</b> ment															. 702
FOREIGN BODIES	IN	THE	E	CSOP											. 703
HEMORRHAGE .															. 704
Treatment	•		•			•	•		•	•	•	•	•	•	. 705
		(	H	AP	TE	R	XΣ	XX.	IV						
			Α	LI	MEI	NT	AT]	O	1						
ECONOMIC IMPOR	TAN	CE C	F	Pro	PER	ΑL	ımı	ENT	ATI	ON					. 706
SELECTION OF I															. 711
RECTAL ALIMEN	TATI	ON													. 727

•

#### LIST OF ILLUSTRATIONS

	ING GE
I.—Types of Stomachs	84
II.—Schematic Drawings, Illustrating Interpretation of Radiograms	90
III.—Carcinoma Involving Esophagus and Cardiac End of the Stomach	<b>404</b>
IV.—Cancer Growing In Scar of Preceding Ulcer	418
V.—Points of Tenderness Present in Abdominal Disease	662
RADIOGRAMS  I.—Displacement of Stomach Occasioned by Posture, and in Part Owing to Bismuth. Prone Position	118
II.—Displacement of Stomach Occasioned by Posture, and in Part Owing to Bismuth. Standing Position, Same	
Patient	118
III.—Cecum Mobile. Gastroptosis	118
IV.—Cecal Stasis	118
V.—Gastric Spasm. Thirty Minutes After Bismuth Meal . I	180
VI.—Gastric Spasm. One and One-Half Hours After Bismuth Meal	180
VII.—Gastric Spasm. Taken On Another Day, Four Hours After a Bismuth Meal	180
VIII.—Unusual Case of Hour-Glass, or Bilocular Stomach 1	180
IX.—Pyloric Stenosis Following Chronic Ulcer	236
X.—Pyloric Stenosis From Ulcer, Causing Dilatation and Stagnation	236
XI-Pylorus Dragged Downward and to the Left 2	236
XII.—Cicatricial Pyloric Stenosis With Dilatation and Bulging of the Prepyloric Region, the Pylorus Thus Being Covered Over	236

xxxii LIST C	)F	ILLUSTRATIONS
--------------	----	---------------

RADIOGRAMS	PAGE
XIII.—Hour-Glass Stomach in a Woman of 50; Result o Chronic Ulcer. Standing Position	f . <b>2</b> 90
XIV.—Same Case, Patient in a Lying Position	290
XV.—Cardiospasm. Enormously Dilated Esophagus, Thirty Years' Duration, Promptly Relieved by Stretching With Sippy's Dilator	
XVI.—Esophageal Stenosis With Moderate Dilatation	452
XVII.—Esophageal Stenosis With Dilatation From Carcinomat ous Infiltration	452
XVIII.—Esophageal Diverticulum	452
XIX.—Cancerous Infiltration at the Pylorus and Greater Curvature	
XXScirrhous Infiltration at Pylorus and Greater Curvature	684
XXI.—Atrophy of the Stomach Following Stenosis at Cardia	684
XXII.—Fish-hook Type Showing Marked Ptosis	684
FIGURES 1.—Stomach In Relation to Heart and Other Viscera	3
2.—The Empty Stomach of a Man Instantly Killed by Falling From a Tall Building	4
3.—Stomach From a Man Who Died Suddenly After a Moderately Full Meal	5
4.—Axes of Upper and Lower Straits of Stomach	6
5.—Longitudinal Section of the Fundus Glands of the Human Stomach	8
6.—Stomach of Usual Form in Relaxation, Showing Location of Important Vessels and Lymphatics	10
7.—Regions of the Stomach	16
8.—Benign Stagnation	64
9.—Pencillum Glaucum, Found in Gastric Contents	64
10.—Gastric Contents From a Case of Carcinoma	64
11.—Sedimented Gastric Contents From a Fasting Stomach	64
12.—The Stomach Tube Described	68
13.—Gastric Ulcer	231

LIST OF ILLUSTRATIONS	xxxiii
FIGURES 14.—Gastritis With Ulceration	PAGE . 233
15.—Old Gastric Ulcer With Induration, Perforation and Localiz Peritonitis With Adhesions to Anterior Abdominal Wall	
16.—Section From a Simple (So-Called Follicular), Erosi of Hayem	on . 240
17.—Section From the Border of Simple Erosion, Shown Fig. 16	In . 241
18.—Section From a Case of Hemorrhagic Erosion of Hayem	. 243
19.—Gastric Ulcer	. 259
20.—Specimen Showing the Pylorus From Case Described	. 281
21.—Hour-Glass Stomach	. 290
22.—Duodenal Ulcer	. 297
23.—Duodenal Ulcer. Located Directly Beneath the Pyloric Ri on Posterior Wall	ng . 299
24.—Perforating Duodenal Ulcer	. 302
25.—Fragments of Tissue Removed From Wash Water in Case Gastric Erosion	of . 374
26.—Einhorn's Gastric Erosion	. 375
27.—Einhorn's Gastric Erosion	. 376
28.—Sieving Pail of Bassler For Lavage Water	. 377
29.—Cancer	. 398
30.—Flat Polyp of the Stomach With Carcinoma Engrafted .	. 399
31.—Beginning Carcinoma of the Stomach	. 400
32.—Adeno-Carcinoma of the Stomach	. 401
33.—Carcinoma of the Pylorus	. 402
34.—Cancer Growing on Ulcer	. 415
35.—Gastric Contents From a Case of Benign Stenosis	. 442
36.—Gastric Contents From a Case of Carcinoma of the Pylorus	s . 442
37.—Leptothrix From the Mouth In a Fresh Preparation Stain With Lugol's Solution	ned . 443
38A.—Long Bacilli From Stagnating Esophageal Contents, in Fresh Preparation Stained With Lugol's Solution, givino Starch Reaction	a. ing . 443

xxxiv	LIST	OF	<b>ILLUSTRATIONS</b>
attract v		OI.	IDDONITULITOIN

FIGURES	PAG
38B.—Long Bacilli From Stagnating Esophageal Contents, in a Fresh Preparation Stained With Lugol's Solution, Giving a Beautiful Starch Reaction	444
29.—Long Bacilli From Stomach Contents in a Case of Gastric Carcinoma, in a Fresh Preparation Stained With Lugol's Solution, Giving a Beautiful Starch Reaction	
40.—Long Bacilli in Intestinal Contents in a Fresh Preparation Stained With Lugol's Solution. Giving a Beautiful Starch Reaction	
1.—Clostridium Butyric (Prazmowski), in Intestinal Contents in a Fresh Preparation, Stained With Lugol's Solution, Giv- ing a Beautiful Starch Reaction	
42.—Gastrectasis With Secondary Emaciation	508
43.—Acute Dilatation of the Stomach From Obstruction of the Duodenum	
44A.—Faulty and Correct Standing Posture With Reference to Intra-Abdominal Pressure	
44B.—Faulty and Correct Standing Posture With Reference to Intra-Abdominal Pressure	
45.—Exaggerated Dorso-Lumbar Curve With Compensatory Exaggeration of Backward Lumbar Curve	
46.—Correct Position	<b>53</b> 4
47.—Absence of Normal Dorso-Lumbar Curve	535
48.—Exercise for Abdominal Ptosis and too Pronounced Lumbar Anterior Curve	544
49.—Exercise for Abdominal Ptosis and too Pronounced Lumbar Anterior Curve	544
50.—Exercise for Abdominal Ptosis and too Pronounced Lumbar Anterior Curve	
51.—Exercise for Abdominal Ptosis and too Pronounced Lumbar Anterior Curve	546
52.—Exercise for Abdominal Ptosis and too Pronounced Lumbar Anterior Curve	547
53.—Exercise for Abdominal Ptosis and too Pronounced Lumbar Anterior Curve	548
54.—Position For Abdominal Massage In Splanchnoptosis	551

LIST OF ILLUSTRATIONS	XXXV
55.—Section Through the Wall of the Stomach at the Seat of Simple Ulcer Which Was Found at the Expense of the	
Mucosa	. 562
56.—Polypoid Gastritis	. 576
57.—The Algesimeter of Boas	. 652
58.—Boas' Point, Represented by Shaded Square at Right of Vertebrae	
59.—Diagram Showing Location of Abdominal Pain in Variou Affections	
60.—Fraction Diverticulum of the Esophagus, Communicating With the Trachea	
61.—Stockton's Combined Stomach Tube and Gastric Electrode	. 676
62.—Einhorn's Cardio-Dilator	. 681
63.—Sippy Dilator	. 687
64.—Cancer of the Esophagus	. 691
65.—Dilatation of Esophageal Veins	. 704



# DISEASES OF THE STOMACH

# CHAPTER I

# ANATOMY AND PHYSIOLOGY

Practically, digestion begins in the kitchen. In the selection and preparation of food, in the dressing and serving of it, are involved great principles which, through psychic and physiologic sides, modify the physiology of digestion.

In the normal individual the first step in digestion is psychic; it begins with the concept of enjoyable eating, with the pleasing sight, smell and taste of food.

Digestion is a matter of hydration of food through the action of ferments in an alkaline or an acid medium, and this is greatly facilitated by the separation of the morsels by mastication. Of late much has been said of that part of digestion which takes place within the buccal cavity, and though its importance has been exaggerated, much needed attention has been directed to the value of very thorough mastication, which is especially requisite as regards carbohydrates and vegetables. Faulty teeth, which render difficult the separation of morsels of food, disturb digestion not only by rendering mastication painful but indirectly by disturbing appetite, innervation and The teeth not only should be sound but properly arranged and the mucous membrane must be healthy so that mastication may be done comfortably. When the secretion of the salivary glands is thoroughly mingled with the aliment, there commences in the mouth the hydration of the amylaceous material, and this continues

for some time after starch has reached the stomach. Deglutition, after the food has left the pharynx, is carried on by the rhythmic peristaltic waves of the unstriped muscle in the esophagus which carry the bolus into the stomach. In the mouth there is the exercise of voluntary motion; in the esophagus involuntary; and in the pharynx a combination of the two. The mechanism of deglutition is especially complicated in the pharynx. That must be regarded as a remarkable contrivance which carries the food alongside and beyond the glottis without embarrassment to the function of the respiratory tract. Disease of the larvnx or other parts of the respiratory apparatus may produce sympathetic disturbance of the motor function of digestion. The normal tonicity of the esophagus depends upon innervation from the vagus. This nerve is stimulated by the act of eating, as may be shown by the effect of eating upon circulation; and derangement of vagus innervation may exercise an unfavorable effect upon digestion. less through the action of the vagus there is proper tonicity of the walls of the esophagus, the rhythmic peristaltic waves become ineffectual and deglutition is impeded.

The lower part of the esophagus is more abundantly supplied with mucus which facilitates the passage of boluses of food at the same time protecting the esophageal wall from irritation. When a bolus is swallowed it passes quickly downward, propelled through the region supplied by voluntary muscle fibers, until it reaches the lower third, where the muscle fibers are of the unstriped variety. Through this portion deglutition is more deliberate, and the rhythmic peristalsis of the esophagus is more evident. When the food reaches the end of the esophagus, the cardia relaxes.

Upon relaxation of the cardia there is a momentary inhibition of the movements of the stomach which assumes a receptive state and passively receives the food. This accounts for the fact that gastric distress accompanied by over-tonicity of the stomach, is usually promptly, although temporarily, relieved by the swallowing of food.

# STOMACH

Shape and Position.—There is a lack of agreement between anatomists and clinicians as to the shape and position of the stomach. When viewed post-mortem, it looks

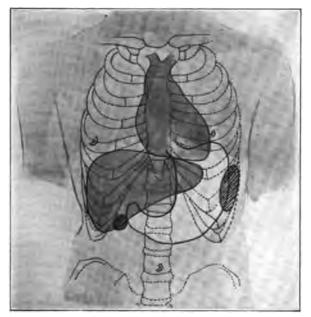


FIG. 1.—STOMACH IN RELATION TO HEART AND OTHER VISCERA.

very different from what is inferred from radiographic shadows in the living, and the contour as seen by the surgeon, when operating, is different from that observed through the fluoroscope during the act of normal digestion. A straight line, drawn through the stomach at the outlet of the esophagus and parallel with the direction of the latter, would divide the stomach into two unequal parts. The cul-de-sac at the left, comprising about one-fourth of the organ, is known as the fundus.

In the normal individual, during the state of physiologic activity of the stomach the fundus lies behind the heart, its superior surface resting against the arch of the



FIG. 2.—THE EMPTY STOMACH OF A MAN INSTANTLY KILLED BY FALLING FROM A TALL BUILDING. The shape of the stomach is exactly as it was when removed at autopsy. Note the muscular development of the pyloric portion. (By Permission of Prof. George S. Huntington.)

diaphragm. From the standpoint of physical diagnosis the fundus appears to occupy the thorax. The left border of the fundus, along the greater curvature, is in relation with the left kidney and the spleen. The cardia is at the left border of the sternum, behind the sixth and seventh costal cartilages and corresponds with the tenth dorsal vertebra. The pylorus, at its superior border, corresponds posteriorly with the second, third or fourth lumbar vertebra and is often partly covered by the liver.

Its center lies at the junction of a line drawn down the right border of the sternum and a horizontal line which varies in different individuals drawn somewhere above the umbilicus. It is located about 3 cm. to the right of the median line when the stomach is full, but when the stomach is empty it is usually found at the median line and perhaps slightly to the left thereof. The upper or lesser curvature is parallel in its upper two-thirds to the



FIG. 3.—STOMACH FROM A MAN WHO DIED SUDDENLY AFTER A MODERATELY FULL MEAL. The shape of the stomach is exactly as when it was removed from the body. The muscular development of the pyloric third is well shown. (By permission of Prof. George S. Huntington.)

border of the sternum, while the lower third bends transversely or obliquely to the right. The transverse portion of the lesser curvature crosses at a point about 4 or 5 cm. below the xiphoid cartilage. The lower third of the stomach during the state of physiologic activity assumes a more or less tube-like form, in the normal individual. Its shape changes in proportion to the tonicity of the stomach.

The position of the lower border of the moderately filled stomach varies considerably in different individuals. Ordinarily it is found from 2 cm. to 3 cm. above

the umbilicus. It is found that in some apparently healthy individuals the stomach occupies a relatively perpendicular position, a position which Holtzknecht considers to be common.

Under such circumstances the lower end of the stomach usually bends somewhat sharply upwards and to the right. As a result, the pylorus is located at the point hitherto de-

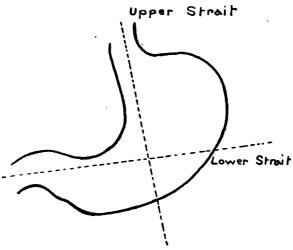


FIG. 4.—AXES OF UPPER AND LOWER STRAITS OF STOMACH.

scribed. Occasionally, the duodenum joins the pylorus at a point below that above indicated, constituting the so-called pyloroptosis. A good idea of the shape of the stomach may be gained by considering the upper and lower axes. The former is determined by extending through the stomach a line drawn down the esophagus; the latter by a line drawn transversely across the stomach from the outlet of the pylorus.

Structure.—The stomach is a pouch of unstriped muscle tissue, the outer fibers of which are arranged longitudinally; beneath these are circular fibers, and innermost of all is a rather incomplete set of oblique fibers. These are

best seen in the upper part of the stomach where they are continuous with like fibers of the esophagus.

Internal to the oblique muscular layer, between it and the mucosa, is the areolar or submucous coat which is richly supplied with vessels and is of such loose structure as to admit of easy folding in all directions.

The gastric mucous membrane is covered with a layer of columnar epithelial cells which are for the most part mucus-secreting goblet cells. The substance of the mucosa is made up of the peptic glands packed closely together, running in a vertical direction and so arranged that two or three unite to form a common tube that opens upon the surface. Owing to the great number of these tubes whose openings thickly stud the folded surface of the stomach, there is seen a resemblance to villi, making up the so-called plicae-villosae. The individual glands extend downward toward the muscularis mucosae, and are divided into a mouth, neck and body. Columnar epithelium lines the mouths of these glands. while the neck, which is the thinnest portion, and the body are lined with the specific secreting cells of the stomach. The layer of cells lining the tube glands is known as the layer of chief cells and these cells are believed to secrete the specific enzymes of the gastric juice. (Heidenhain.) Outside these cells and thus more deeply situated there occur from point to point single, separate, oval, spherical or angular cells, much larger than the chief cells, and of a granular and opaque These are known as the parietal cells. appearance. (Heidenhain.) They are believed to secrete the hydrochloric acid. These cells, more numerous in the neck than in the fundus of the glands, have a densely reticulated protoplasm. The chief cells form the largest portion of the gland. They are round or cubical, their form and size depending on their functional activity. During a period of fasting and at the beginning of digestion

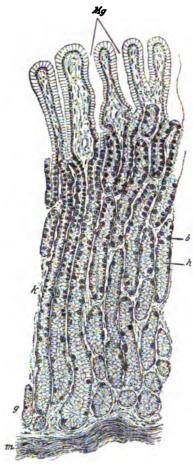


FIG. 5.—LONGITUDINAL SECTION OF THE FUNDUS GLANDS OF THE HUMAN STOMACH. b, parietal cells; g, fundus of the gland; h, chief cells; k, body; l, neck of the gland; m, muscularis mucosae; mg, gastric crypts. x85. (after Kölliker). (By permission of Dr. Ferguson.)

they are large, while after digestion has proceeded for a certain length of time they become much smaller. In the fresh condition they contain numerous highly refractive granules, which, as in other glands (pancreas, parotid, etc.), disappear in the outer zone of the cell during secretion. These granules are supposed by most authorities to consist of substance, pepsinogen, which is converted into pepsin.

The pyloric glands differ somewhat in structure from the peptic glands which belong to the larger area of the gastric lining. The duct of each pyloric gland is longer than the ducts from other parts of the mucosa, although lined by the same kind of epithelium. Two or three gland tubes unite their thin necks to form the

duct. The gland tube itself is convoluted and slightly branched.

The parietal cells in the neck of the gland are usually numerous, and may lie in rows like the chief cells. are generally only sparingly present in the gland body. Here they are pressed out by the chief cells against the membrana propria so that they seem to be at the periphery of the tubule. The parietal cells are round or polygonal, finely granular cells, containing one or two spherical nuclei. They are smallest after digestive activity and increase in size before and during digestion. the fresh state they are clearer than the chief cells, while in fixed preparations they are much darker and less clear than these. The transparent substance between the meshes of the cell reticulum increases in amount during secretion. Those parietal cells which are not situated directly on the gland lumen are connected with it by a secretory duct, which breaks up into a number of secretory capillaries. These surround the cell like basket work, and also project into its interior. The cells which are situated along the edge of the gland lumen do not possess a duct, as their secretory capillaries empty directly into the gland lumen.

In that region where the pylorus merges into the larger portion of the stomach, the secreting glands take on a form intermediate between the pyloric and the peptic glands.

Lymphatics.—Bedded in the mucosa are many lymph follicles, glandulae lenticulares, and these are especially numerous in the pars pylorica where they also occur in groups, the glandulae agminatae.

A network of lymphatics is found at the body of the glands leading into freely anastamosing lymphatics which run longitudinally between the glands and approach the surface; another plexus of lymph vessels belongs to the submucosa.

The distribution of lymphatic tissue is abundant, not only in the pyloric region, but along the lesser curve of the stomach, especially towards the pylorus. There are many lymph-paths which communicate with the lymphatic plexuses in the submucous coat, and which pass to the superficial and deep sets of lymphatic glands situated along the curvatures of the stomach.



Fig. 6.—Stomach of Usual Form in Relaxation, Showing Location of Important Vessels and Lymphatics.

The Blood Supply.—Ensheathing the gland tubes is a longitudinal network of capillary vessels derived from the arteries of the submucosa. Along the surface the capillaries are especially dense and are distributed in a horizontal layer. These vessels have a distinct supply as have those of the muscular coat and the muscularis mucosa.

The vascularity of the stomach is remarkable. Its arterial supply is derived from the gastric, the vasa brevia, the right and left gastro-epiploic and the pyloric

arteries; along the lesser curve, the gastric anastamoses with the superior pyloric branch of the hepatic artery, thus forming the coronaria ventriculi.

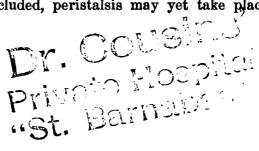
The venous radicles are fewer but much larger than the arterioles; they form trunks which empty, some directly into the portal, others into the splenic vein. The small pyloric vein is regarded as an important surgical landmark and is thus described by Moynihan. "It runs generally a little to the gastric side of the pylorus. The vein runs upward from the greater curvature, and is thick and short. It may oftentimes be met by a smaller vein descending from the lesser curvature; usually the two do not meet in a visible anastomosis." This vessel is supposed to mark the juncture of the pylorus with the duodenum.

The Nerve Supply.—The innervation of the stomach is both intrinsic and extrinsic.

Besides Meissner's plexus, the intrinsic innervation depends upon Auerbach's plexus, extending itself between the outer longitudinal, and the inner circular, unstriped muscle layers of esophagus, stomach and intestine.

The extrinsic innervation is from three sources: first, from the bulb, reaching the stomach through the vagi; second, from the sacral nerves; third, from the sympathetics.

The nerves of Auerbach's plexus, constituting the intrinsic innervation, are spoken of as the "myenteric plexus," and are called by Cannon, the "myenteric reflex." These nerves appear to inaugurate the peristaltic waves in the esophagus and intestine as well as in the stomach; however the rhythmic peristalsis is not limited in origin to the action of the "myenteric reflex" for, when this is excluded, peristalsis may yet take place,



although somewhat tardily and incompletely. It is shown that the effect of the "myenteric reflex" is to produce a zone or ring of contraction, immediately below which there occurs relaxation of the muscle layer, producing a ballooning of that portion of the stomach.

In order that the "myenteric reflex" should act, it seems to be necessary that the walls of the stomach should be in a state of tonus. When they are in this state, the stomach contracts upon its contents. The arrangement of muscle fibers is such that there is greater contractility exercised in the neighborhood of the pylorus than in that of the cardia. When for any reason there occurs, at a definite zone of the stomach, a degree of tension sufficient to give rise to stretching of the muscle fibers at this zone, the "myenteric reflex" is excited at the ring of contraction and subjoining relaxation appears.

This stretching seems necessary in order that the resulting contraction should arise. It will, therefore, be seen that in order to execute its function of producing rhythmic peristalsis, the "myenteric reflex" must have already provided a tonicity of the gastric walls, a general contraction that will cause stretching at some particular zone of the stomach greater than at other zones, for otherwise the stimulus to the reflex would be everywhere alike, and no ring of contraction would follow. To understand why stretching is exercised at one point more than at other points in the circumference of the stomach, it is necessary to recall that the muscle layers at the fundus are comparatively thin and that they progressively increase in thickness to the pyloric sphincter. With a given degree of intragastric pressure, a greater pull would be sustained by the thin muscle fibers of the fundus than by those of the denser layers toward the pylorus.

Now there is a relation between the existing general tonus of the gastric walls and the intragastric pressure. With too great pressure in proportion to the tonus, or with too little pressure, the reaction causing the peristalsis does not occur. With the proper degree of intragastric pressure there will arise at some particular zone of the stomach just the needed stretching in proportion to the tonus, to excite the reflex and to produce the contracting wave. That the peristalsis proceeds onward rather than backward is explained by the fact that the contracting ring puts a stretch upon the circle of muscle fibers immediately below, thus stimulating a new zone to contract, and so on until the pylorus is reached.

The region behind is in a state of tonus sufficient to render it refractory to the stimulus of the intragastric pressure. The region of the stomach at which a peristaltic wave will start can not be determined. It arises sometimes near the pylorus, but at a different stage of digestive activity it will arise midway between the pylorus and cardia, or at some other point. This question of gastric peristalsis is of the utmost importance, not alone in the interest of digestion, but in the explanation of symptoms when the stomach is acting out of harmony.

It will be observed that the question of tonus acquires, from this point of view, a fresh importance. How is it that tonus arises? In what way is it induced and how is it inhibited?

It has been experimentally shown that among other functions the vagi have that of producing tonus in the stomach, as in the esophagus. When stimulated, the bulbar impulses pass through the vagi to the stomach

and, after a brief phase of relaxation, the organ becomes tonic, remaining so for a considerable period of time.

The vagus, therefore, while it does not directly induce the peristalsis, prepares the way for it; it contributes the factor of tonicity. The stomach contents contributes by making possible intragastric pressure; the interaction of internal pressure and tonus, excites the "myenteric reflex," and the peristaltic ring.

The complicated mechanism of gastric motility needs yet further description. There is another source of tonicity besides that coming from vagal action.

When the stomach is released from all pneumogastric influence through careful section of all fibers of the vagus, there is here, as in the case of the esophagus, a period during which the organ is in a state of complete relaxation; food introduced lies unmoved.

After the lapse of two or three days, peristalsis may be developed by the introduction of food, sufficient to cause slight stretching from intragastric pressure. Eventually these peristaltic waves become larger and stronger. This property of automatic peristalsis on the part of the gastric muscle, when subjected to a stretch. closely resembles that which occurs under normal conditions through the agency of the vagi. The function of the vagi, therefore, is that of promptly inducing a muscular tonus in the walls of the stomach and this tension acting upon the stomach contents exerts a stretching effect upon the muscle fibers and, while this may occur without the action of the vagi, it is not what ordinarily happens except when the digestion is well started. If at this time the nerves are cut, the peristalsis will continue undisturbed through its own automatic mechanism. The motor function of the vagi, then, appears to be of advantage in producing an early establishment of peristalsis, although the property of those nerves is not requisite when active wave movements are thoroughly established. (See Cannon, Mechanical Factors of Digestion.)

When a bolus is swallowed and passed down the esophagus through the action of the physiologic power just described, it is received, upon reaching the cardia, by a state of relaxation which suits its ready entrance into the stomach. This inhibitory action also appears to come through the vagus and is not displayed when the vagus is severed. But for the most part, the motor inhibition of the stomach is conveyed through the splanchnics. These sympathetic nerves appear to possess the office of antagonizing the vagal or autonomic nerves. is interesting to know that the stomach when stripped of all nerve connections is able, through its automatic mechanism, to carry on its motor function. When either the autonomic (vagal) or the sympathetic (splanchnic) nerves are alone removed, the other being left in operation, the motor function is more embarrassed. lightens us on the clinical fact that whenever either the vagal or sympathetic nerve is over-stimulated there results a disturbance of digestion. From another point of view this enlightens us as to the process at work when psychic stimulation on the one hand and abdominal irritation on the other, produce their well-known effects upon digestion.

Physiologically speaking, the stomach is divided into two parts; the larger, that towards the cardiac end, serves as a receptacle for food, which is held under steady pressure through the tonicity of the fundus. But little active digestion occurs at this end of the stomach except that which is effected by the action of ptyalin; for a considerable time after ingestion, this continues its amylolytic power, especially in the center of the food mass. The second and lesser part of the stomach, that towards the pyloric end, soon after ingestion of food, takes on an active peristalsis. Its contents soon becomes highly acid, not alone from the secretion of its own glands, but from the secretion of the cardiac half which is brought onward to the pyloric region through superficial peristaltic waves. These occur along the greater (cardiac) part of the stomach, sufficient in extent to carry the secretion onward, but not sufficient to press the secretion into the mass of food resting in this pouch. As a result, only the more superficial layer

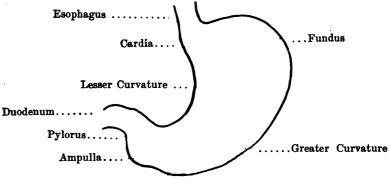


FIG. 7.—REGIONS OF THE STOMACH.

of food in the fundus of the stomach is at this time attacked by the gastric secretion; and this secretion, being carried onward by the small peristaltic waves, reaches the pyloric extremity where it accumulates in considerable amount. This abundant gastric juice, mixing with the food through the assistance of the strong prepyloric peristalsis, rapidly dissolves the soluble part, which first softens, then liquifies and in this condition is prepared for passage through the pylorus.

Contrary to the belief following the observations of Beaumont, more recent investigations show that there

is in a general sense no current of contents through the stomach during gastric digestion. The food in the cardiac pouch of the stomach lies practically motionless, except in that region where it merges into the prepyloric region. There is, however, an active motion of the contents in the pyloric vestibule, and this is most marked as the pylorus is approached. The constricting band-like fibers in the antrum pylori contract to such an extent that temporarily, the lumen of the part is nearly ob-This heightened pressure of the contents in the prepyloric space is relieved by the backward escape through the narrow opening left by the incomplete closure of the contracting ring. Through this small opening the contents is squirted backwards as a central stream. against the mass of food held under tension in the pouch of the stomach. To accomplish this, it is obvious that the intragastric pressure during peristalsis is greater at the pyloric than at the cardiac end.

The studies of Cannon and Blake show that the solid particles are propelled backwards by this squirting process, and that they do not escape through the pylorus except at the end of gastric digestion. It will therefore be seen that the first half of the stomach has the office of holding under tonic pressure the greater part of the meal while amylolytic digestion is in process; and that the second part of the stomach accumulates the gastric juice and, by the aid of its deep peristaltic waves, mixes the chyme, softens the solid particles thus reached, discharges through the pylorus the soluble portions, and rejects, by a central stream, the more resisting particles to be further attacked. As time elapses, the gastric secretion slowly permeates the food mass, remaining in the reservoir of the stomach, and chymefaction thus proceeds slowly from without inward. Even with a semifluid meal these facts hold true although to a less extent. Half an hour or even longer is required for the central mass of a solid meal to become acidulated. In normal digestion, the cardiac opening and, during most of the time, the pyloric opening remain closed; the pyloric sphincter relaxes and permits the escape of liquid contents under the control of the duodenal reflex, as described by Starling. That is to say, when a small amount of acid chyme passes through the pyloric opening into the duodenum, that organ through a nerve reflex causes contraction and closure of the pylorus. Upon the neutralization of a portion of chyme received by the duodenum, the pylorus again relaxes, another portion of chyme is discharged into the duodenum, the reflex is again awakened, thus closing the pylorus, and so on, until the stomach is empty. Provided there is no abnormal irritation to disturb the harmony of gastric tonicity, peristalsis, duodenal reflex and pyloric opening and closing, the stomach will empty itself completely after a given length of time, four or five hours after an ordinary meal. The ingested solid fragments are at last discharged as a result of the tonic pressure from behind and the gradual subsidence of the active peristalsis in the prepyloric region and, ultimately, the relaxation of the pylorus. When at rest, the stomach is closed at either extremity, thus preventing the upward escape of a certain amount of gas that is always present and the retrograde discharge of duodenal contents through the pylorus.

Radiographic studies show that a considerable volume of gas is normally present in the stomach, and is represented by a clear space, resembling a dome, under the arch of the left diaphragm. The shape of the stomach undergoes remarkable variations during gastric digestion. There is but slight evidence of this in the upper, hopper-like pouch, but in the second half of the stomach the contour changes sometimes assuming the form of a tube, in shape resembling an intestine, sometimes serrated by small peristaltic waves and at other times appearing almost divided through the constriction produced by staltic contraction of unusual intensity. (Radiogram XXII.) From radiographic study it is difficult to escape a false impression as to the shape of the stomach during active digestion. The instantaneous impression of a single radiogram is insufficient. A better conception of the gastric motility can be obtained by a series of plates or, better still, by observations with the fluoroscope.

#### GASTRIC SECRETION

The gastric juice is secreted by the glands of the gastric mucosa. Chemically, it consists of free hydrochloric acid, water, ferments, zymogens, mineral salts and mucus. The hydrochloric acid is secreted by the parietal cells, the pepsin and rennin by the chief cells, the mucus by the goblet cells of the gastric mucosa. The ferments are present as zymogens which are transformed into ferments by the action of free hydrochloric acid.

The gastric juice is a clear, colorless fluid containing 0.3 to 1 per cent total solids, with an acidity equivalent to 0.48 per cent hydrochloric acid. (Starling.) That which is secreted from the fundus is highly acid, while that coming from the pyloric portion is practically neutral.

The secretion of gastric juice results from stimuli of different kinds. The first to act arises in psychic centers and is excited not only by the smell and taste of food, but by the sense of hearing or any impulse that suggests the idea of eating, as shown by Pawlow. These impulses are conveyed to the stomach through the vagus nerves. This, however, does not account for the whole of gastric secretion. Aside from that which is secreted as a result of vagus stimulation, a certain proportion of gastric juice is excited by the presence of food in the stomach. It has been shown that certain foods evoke this secretion while others in this respect are ineffective. Meat, and especially meat broth, or extract, quickly excite the secretion, yet the introduction of the white of egg, starch or bread is inoperative. However, if bread be mixed with gastric juice and allowed to digest for a time, it excites even more active secretion than bouillon. phase of gastric secretion does not depend upon stimulation of the vagus, but upon some local action of the food upon the gastric mucosa. Popielski believed that this resulted from a local nervous reflex residing in the gastric mucosa. Edkins, however, has shown that this portion of the gastric secretion may be accounted for in the action of a "gastric hormone." He was able to procure this substance by boiling the pyloric mucous membrane with acidulated water and by other methods. When this substance was injected into the circulating blood it led to the active secretion of acid gastric juice. It was not possible to procure this substance from the mucous membrane of the fundus, but only from that of the pyloric region. It is established, therefore, that gastric secretion in part depends upon the mechanism of a chemic signal, and only in part upon autonomic or vagus stimulation. Whether there also exists a local reflex secretion of gastric juice, as believed by Popielski and Pawlow, is perhaps not fully determined.

In the order of development, the hormones appear to have been the earliest provision of nature for coördi-

nating the processes of digestion. By means of these substances, without the intervention of the nervous system, the secretion of special ferments are excited, or those already secreted may be activated and, by the action of other hormones, the motor activity of the stomach and intestines is initiated. As a second step in development and as an accessory means of control and coördination, there appear the autonomic and sympathetic or ganglionic systems of nerves. These are endowed with a certain unconscious control both of secretion and motion and doubtless serve to economize energy and expedite the performance of function. Later there came into the field the cerebro-spinal nervous system and hence the introduction of intelligence or conscious effort in the matter of digestion.

The harmonious working of these various mechanisms, in order that there may be a more efficient apparatus for the use of the commissary of the organism, is a marvelous example of adaptation. We find that some organs in the performance of function utilize these mechanisms in a selective manner, sometimes perhaps substituting one for the other, as for instance when the internal gastric secretion fails we may suppose that the work is carried on by the autonomic nervous system.

In the stomach we find the fundus, a comparatively late accession to the organ, relatively more dependent upon nerve control than the pylorus, even having an element of cerebro-spinal or voluntary nerve control, a feat that appears to be beyond the powers of other portions of the gastro-intestinal tract except the rectum. Nevertheless, the pylorus is the more active part of the stomach; it is related to the fundus as in the fowl the gizzard is related to the crop.

While the pylorus is somewhat dominated by the special secretions, it is also under the jurisdiction of the nervous system.

Normal gastric digestion proceeds as follows: With the first taste of food and even before this, with the mere announcement of a repast, with the sound or smell of its preparation, there promptly begins the secretion of gastric juice and of the salivary ferments. These are still further stimulated by the mastication of food and the thorough intermixture of the saliva. Thus amvlolvtic digestion begins in the mouth and continues thereafter in the stomach. A small amount of the more soluble matter is carried to the further extremity of the stomach. where it serves to excite the secretion of the gastric hormone, and tonicity of the gastric walls. As a bolus of food passes through the cardia into the stomach, there is a transient relaxation of the gastric tonus which promptly reappears, thus holding the ingesta under pressure in the proximal part of the stomach. Here it remains for a considerable time with little movement and. with the exception of a certain amount of gas, it fills the reservoir or pouch of the stomach. These conditions are accomplished whether the repast be moderate or large, as the tonic gastric walls contract about the stomach contents so that the temporary capacity is suited to the amount of the ingesta. The acid secretion of the fundus is carried along the lining of the stomach by superficial peristalsis towards the pylorus where it accumulates and through the action of strong and deep peristaltic contraction, it is brought into contact with the food so that proteid digestion becomes active. Meantime, the starch digestion proceeds in the center of the ingested mass which yet remains in the pouch of the stomach.

A more definite conception of the mechanism of digestion may be obtained by considering the somewhat recent studies of the action of the autonomic division of nerves and of the antagonism which appears to exist between the action of these nerves and of those that are more strictly classed as sympathetics.

The subject is more fully discussed on page 104.

# CHAPTER II

#### EXAMINATION OF THE PATIENT

We must listen to the recital of the complaints of the patient without the preconception that their source is in the stomach. The object is primarily to make a diagnosis and in this, gastric pathology may occupy unexpected relations.

Within limitations the patient should be allowed to tell his own story before he is subjected to interrogations, for in this way there may be obtained quickly important information regarding his general tendencies.

By his conversation and manner, the alcoholic is recognized; the psychopathic patient betrays himself, as does the victim of introspection, or of too vivid imagination, or of neurasthenia.

Due value must be given to the family history. The prevalent belief that dyspepsia depends upon hereditary influences has some justification. Inquiry should be made as to the existence of mental disease, neurasthenia, alcoholism, tuberculosis, arthritic affections, disease of the liver, and especially as to general heartiness and robustness in the parents.

### PERSONAL HISTORY

In addition to being allowed to give a description of his various illnesses, the patient should be led to describe the important events of his life. It is often an assistance to require the patient to write a brief autobiography. Was there a happy and healthy childhood? Gastric disturbances of middle life sometimes begin in infancy or may develop at puberty or result from unhygienic student life, or from "child labor."

Hardships in child life sometimes leave their impress upon the health, and the custom of sending children long distances to school in the country with scanty and improper lunches is responsible for some instances of digestive disturbance in youth. Morbid appetites and habits of eating which are exhibited in some children may give rise to aggravated irritation and over-distension of the stomach. It is a lamentable fact that some children pass through attacks of appendicitis or gastroenteritis with the most inadequate and haphazard care, often without a physician's advice, thus initiating a morbid condition which may have a lasting influence upon the digestive ability of the individual.

Is there a history of preceding illnesses in adult life and do any of these appear to bear a definite relationship to the dyspeptic symptoms? For instance, typhoid fever or its sequelae, may have been the beginning of motor or toxic disturbances, and so renal or biliary lithiasis, neurasthenia, malaria, pulmonary or cardiac disease, and many other affections may bear an unsuspected etiologic relationship to a stomach disease.

The item of change in residence or occupation may put us on the trail of the disturbed physiology; for instance, leaving an active, outdoor life for a sedentary, indoor occupation is frequently of importance; and, on the other hand, in those who have grown up with limited exercise and abruptly undertake work that necessitates severe organic strain and over-exertion, the digestion is likely to fail. Special vocations should be considered. Office employés, professional men and stu-

dents frequently suffer from having an appetite out of proportion to their physical activity, and as a result develop over-repletion, constipation, or metabolic disturbance. Agriculturists may suffer from a lack of variety or quality of food usually because carelessly prepared, and artisans or railway employés from hurried and irregular meals, or dependence upon the "dinner pail." Seamstresses, tailors, especially when working at home, are harmed by long hours and by faulty posture, resulting in derangement of appetite, anorexia and insufficient nourishment, while lead workers and those exposed to arsenic, dust, etc., suffer especially from deranged digestion. Society women pay the penalty for protracted sessions at bridge, afternoon teas, frequent indulgence in sweets and the adoption of the automobile to the exclusion of walking, by resulting headache, constipation, and gastric irritability.

The habits of the patient should be investigated not only as to the use of alcohol, tobacco, tea, coffee and other stimulants, but also as to the hours of meals, and the quantity and quality of the food taken. The honestly recorded description of the several repasts of the patient and of the nibbling between meals is often a revelation. The quantity of fluid ingested with meals and at other times, the amount of salt and other condiments taken, the stimulation of the appetite by cocktails, hors d'œuvres and bitters, all are important. It is well to put a formal set of questions as to eating.

Is the appetite good? Is the food enjoyed, or taken as a matter of routine while the mind is preoccupied? Does the appetite suddenly fail or does it increase with eating? Is there abundant salivation, or is there a desire for drink with meals?

Immediately after eating, is there a sense of comfort,

or is eating followed by local or general uneasiness or distress? Is it followed by eructations of gas, by regurgitations of food? Is there heart burn or sour stomach?

If epigastric pain or distress occurs, does it seem to result from eating, or is it relieved by taking food? How long after eating is it before the pain or distress comes on? Are there hunger and pain three, four, or five hours after meals and do they cease upon eating? Great exactness in answering these questions should be required.

Is there nausea? Is it produced by eating? If present is it made worse by eating? Is it relieved by eating? Does it appear two or three hours after meals? Is it accompanied by eructations or regurgitations?

Is there vomiting? If so, is it before breakfast, directly after meals, or long after meals? Is the material vomited composed of the food that has been taken at the last meal, or are there remnants of preceding meals? Is vomiting frequent or infrequent? In quantity is it large or small? Is it intensely acid to taste, is it bitter, or has it merely the flavor of the food eaten? the vomited matter simply composed of liquid, without containing food? If so, is it tasteless, bitter or intensely acid? What is the color of the vomitus? Does it contain blood or mucus? Is it the color of strong coffee or of coffee grounds? Is it certain that the ejecta is through actual vomiting, or is it merely brought up by regurgitation, mouthful by mouthful? Is the vomitus ever surprisingly great in amount? Are there frequent efforts at vomiting without the discharge of stomach contents?

Is the act of vomiting painful? Is the desire to vomit irresistible or is it premeditated and assisted?

Does the vomiting relieve pain? Does the vomitus consist of mucus and nothing else? Is it yellow or green in color? Is it preceded by nausea? Is it followed by the desire for food?

On the matter of regurgitation, direct questions should be asked as follows: Is the quantity very small, merely sufficient to reach the mouth, or is it considerable? Does the regurgitation come on very soon after eating? Has it the odor of stomach contents, or is it returned precisely as it was taken? Are the regurgitations preceded by heart burn or sour stomach? Are the taste and odor disagreeable? Is the regurgitation accompanied by eructations of gas or belching? Is the act usually one of belching, with an occasional regurgitation? Is the food brought up from the stomach voluntarily? Is the food remasticated and reswallowed, or ejected from the mouth? Is the regurgitation accompanied by an increased flow of saliva?

Is eructation noisy, explosive and oft repeated, or is it silent and at comparatively long intervals? Is the escape of gas under control, or quite involuntary? Is it preceded by a sensation of fullness or gastric tension, and is this relieved by the discharge of gas? Are regurgitations or eructations induced by the taking of certain articles of food only, or do they ensue regardless of the character of the meal? Are these symptoms especially troublesome during a period of constipation? Is gas raised from the stomach especially under the influence of excitement, worry or other nervous state?

Is constipation continuous or occasional? How long has it continued? Does the stomach trouble appear to depend on constipation? Is the dyspepsia relieved when the bowels are active? Are purgatives habitually taken? If so, of what variety, and how long continued? Does

intestinal gas cause disturbance? Is the gas discharged, or does it merely cause abdominal disquietude? Is the discharge of intestinal gas under easy control? What is the character of the stools as to quantity, appearance and frequency?

Stools.—Is there diarrhea? Is it persistent, or does it come on occasionally? Is undigested food found in the stools? Do they contain blood? If so, does this come in the early or later part of the stools, separate from the stools, or mixed with the stools? Are there occasional discharges of dark-colored material, fluid or semifluid, the color of coffee grounds?

Is mucus found in the stools? If so, is this present as a coating to a formed stool, or is it separate and in masses? Are the stools sometimes composed merely of mucus? Does it sometimes take the form of shreds or strings, and is it sometimes blood-stained? Does constipation, rumbling noises, or abdominal pain precede the passage of a quantity of ropy mucus?

Are the stools ever unexpectedly abundant? Are the formed masses ever of large size, or are they narrow and ribbon-like? Is the stool sometimes composed of a granular substance, or small masses the size of peas or beans? Is there a discharge of intestinal sand which precipitates in water? What is the color of the stools, especially are they very dark or very light? When they are mixed with water, do particles rise to the surface and suggest the presence of grease? Are the movements of the bowels painful either in the abdomen or in the rectum or at the anus? Are the acts of defecation followed by a sense of exhaustion or nervousness?

Investigation should be made concerning the relation that exists between the digestive and the nervous systems. Is there insomnia after a full evening repast, or does eating at night produce sleep? Is there a feeling of faintness and debility on waking in the morning which is relieved by eating? Is the breakfast followed by a state of mental dullness, and general indisposition, or does it give rise to excitement, nervousness and irritability? Is the *sleep* calm and uninterrupted, or are there hours of restlessness, periods of nightmare, or waking with a sense of fear?

Headache is to be studied in its relation to meals, to abstinence, to constipation, to diarrhea, to occurrence in morning or evening, and as to its location and character. Indigestion with gastro-intestinal atony may be induced by sinusitis with imperfect drainage, and this is usually accompanied by supra-orbital headache on the affected side which radiates to the corresponding occipital region. The patient with eye-strain often wakes with a headache, usually hemicranial, which may pass off after breakfast. At other times it is more noticeable after using the eyes in reading, and may develop into a sick headache, with paroxysms lasting for several hours, and even days, to be succeeded by a period of calm without pain. It is remarkable that eye-strain producing headache is less likely to be associated with dyspepsia than when not so associated. Periodic stomach disturbance often depends upon a functional derangement accompanying the catamenia. Inquiry should be directed as to the effect of mental activity upon the digestion, and we should learn whether distress follows emotional excitement or mental strain, as from too close application.

# PHYSICAL EXAMINATION

Before taking up the study of the digestive apparatus one should make a complete physical examination. To

omit this from routine practice is to court misconception and disappointment. In beginning the physical examination, one should consider first the general conformation or decubitus of the patient.

# INSPECTION

With the chest and abdomen exposed, the patient should be inspected first in the upright and then in the horizontal For the purpose of the latter, he should lie upon a cushioned table about three feet high, which may be easily turned so that the light may fall upon the surface of the body from different angles. That is, light should be made to fall from the direction of the left shoulder, then from the side. Standing at the foot of the table, the physician observes the abdomen, while the patient lies with the limbs extended and the body fully relaxed. movements of the abdominal walls during slow, deep diaphragmatic respiration should be noted, which may disclose that there is a lack of symmetry in the two sides, or that the movements of the abdominal walls are at some point impeded or retarded. Attention should be directed especially to the region beneath the costal arch. A prominence under the right costal border may indicate hepatic enlargement; under the ensiform, disease around the lesser curvature of the stomach; and, under the left costal border a dilated, distended, or displaced stomach, or disease of the gastric parietes. In many cases by careful observation, we may form a surprisingly clear notion of the position of that portion of the stomach not cov-This is especially true when there ered by the ribs. exists an over-tonic state of the gastric walls. When there is marked gastric peristalsis, such as is occasioned by pyloric stenosis, the rhythmical peristaltic waves may be seen to pass from the region of the fundus toward

the pylorus, followed in some instances by waves of antiperistalsis. Such results are not obtained as frequently as might be for several reasons: first because the examination is made too hastily, time not being given for deliberate inspection of the abdomen; and, second, because the examination is not made when the stomach is actively working. In other words, the patient should be given a hearty meal an hour before inspection is practiced, and again examination should be made during fasting hours and after the stomach has been emptied by lavage. Further information may be obtained by inspection after the stomach is moderately distended with gas. It is inadvisable to distend it fully with water. A tumor at the pylorus will be found to change its position upon distension or upon emptying of the stomach. For that matter it should be remembered that when the stomach is heavy from food or water, it normally occupies a lower place in the abdomen than when empty or containing a moderate amount of gas. Variations in the position of the organ may be produced through perigastric adhesions that limit its movements. Inspection of the upper part of the abdomen may disclose a movable kidney, an enlarged or displaced spleen, or liver, a tumor in the transverse colon, or an enlarged and indurated pancreas and any one of these may suggest a tumor or deformity of The lower portion of the abdomen should the stomach. also be carefully inspected, amongst other things, for the evidence of unusual pulsation of the aorta, for prominence produced by gastroptosis, disease of the colon or mesentery, enlargement, distension, or a state of overtonicity of the cecum, for intestinal peristalsis and for the general condition of the abdomen. Over-distension of the intestine, or the distension of one part as compared with another, the probable amount of omental fat. and the tonicity or laxity of the abdominal wall should be noted. In the person with good abdominal tonicity, there is relatively but little change in appearance with change in posture; whereas, with a lax wall the abdomen falls through gravity from side to side, suggesting a half-filled sack; and, with the patient lying upon the back, the abdomen is flattened at the median line, and bulges outward at each side greatly changing the outline of the body; when the patient is in the upright position, the abdominal contents causes protrusion just above the pubes.

#### **PALPATION**

In the physical examination of the abdomen, the importance of the method of inspection is secondary only to that of palpation. In practicing the latter, relaxation of the abdominal wall is of prime importance, to secure which it is necessary to induce confidence and a state of calm in the patient. Even under these conditions some individuals are unable to relax the abdomen sufficiently for satisfactory palpation. Requiring the patient to draw up the knees is a conventional plan but it usually yields poor results so that in general it is best to have the limbs extended. The patient should be told to relax the abdomen and not to flinch upon pressure. This he often fails to understand. If instructed to place his own hand upon the abdomen and to secure relaxation to his own touch, the patient may be taught proper co-Often it is well to instruct the patient to press operation. the head backward firmly upon the pillow, and to breathe slowly and quietly. In important cases in which proper relaxation is difficult to secure, the continuous application of hot abdominal stupes is advisable; this may be practiced for hours and even days preceding an examination. Where time is important, the patient should be immersed at full length in a tub of hot water, and palpation practiced under these conditions. In the majority of cases, a satisfactory result is obtained without this trouble. A dusting powder sprinkled over the surface is an aid to delicacy of touch and lessens the susceptibility to muscle spasm on the part of the patient.

There are two methods of palpation, the superficial and the deep. The former of these should be practiced first. In practicing superficial palpation, one should employ the under surface of the fingers, avoiding the palm of the hand, stroking the abdomen slowly and gently from above downward. No attempt should be made to dip deeply. It is sought to learn thus the relative tension and resistance of the abdominal surface and nothing more. It is surprising how much may be discovered by this method when once the art is thoroughly acquired. With deep palpation, one ordinarily begins by employing the palms of both hands simultaneously; the left being used to support the abdomen and to provide counter pressure, while with the right, the entire surface of the abdomen is gone over, area after area, pressing the fingers downward from time to time, so that deepseated resistance may be appreciated. In the region of the stomach one should use both hands. The examiner standing at the side of the patient and facing him, should rest the right hand along the left border of the abdomen just in front of the free ribs while the left hand makes counter resistance in the region of the pylorus. With the right, a forcible push should impel the gastric contents towards the pylorus, and a similar well-timed maneuver with the left hand should force the contents backward towards the fundus, thus producing succussion or clapotage. The results are facilitated by introducing both air and fluid into the stomach, as a preliminary to the practice of palpation. The success in palpation depends largely upon the movement of the viscera during deep abdominal or diaphragmatic breathing. A useful maneuver consists in applying the hands side by side upon the abdomen making but moderate pressure, moving the fingers slightly from side to side and pressing the tips of the fingers downward at the right moment in the endeavor to detect the border of the moving organ or tumor, as it slips upwards or downwards under the abdominal parietes.

Deep Finger Palpation.—In practicing deep palpation, the most nearly complete relaxation possible of the abdominal wall should be obtained by resort to such expedients as best suits the case. Sometimes with the knees flexed, sometimes extended, the patient lies upon the back, side, or in the semi-prone position, whichever gives The conscious cooperation of the pathe best results. tient is, of course, important when it can be secured, and as before stated, assistance may be had from the previous application of hot fomentations or by immersion in a hot bath. Two or three fingers of the right hand should be pressed deeply into the abdominal wall, the attempt being made to feel into the deepest parts of the cavity. Time should be taken carefully to go over the entire abdomen, pausing from time to time to obtain the additional information which may come from the moving of the viscera while the patient breathes deeply. sistance may come from the use of both hands in the following manner: The palpating fingers of the right hand are laid upon the abdomen and the corresponding fingers of the left hand are placed upon these near the nails. Pressure being exerted by the left hand, the fingers of the right are thus forced inwards: this sometimes

results in securing at once a deep pressure and a finer sense of touch than with the right fingers unassisted.

The theory upon which this is based is that while the muscles of the fingers are rigid with the effort of pressure, they lose correspondingly in sensitiveness of touch; the above practice obviates this disadvantage. For practical purposes there is little in the method, and it will rarely be used by one who is skillful with his hand.

The Lower Border of the Stomach.—By a special method of palpation, which amounts to conjoined percussion and palpation, we may outline the most dependent portion of the stomach. Using the right hand with fingers slightly flexed, beginning from below and proceeding upwards, the fingers are quickly pressed into the abdominal wall, thus displacing the fluid contained in the stomach. The displacement conveys to the fingers a peculiar sensation, sometimes accompanied by a slight splashing sound, which enables one to determine whether the impulse is made over the stomach or the neighboring viscera. If the attempt is not at once successful, the patient should drink a large glass of water after which the palpation should be repeated.

The above method is practically that introduced by Obrastzow, which is thus carefully described by Cohnheim. "The patient lies flat on a reclining chair with the upper part of the body slightly raised, and is given one or two glasses of water. The examiner should place on the epigastrium of the patient the fingers of his right hand, spread out claw-shape, and should palpate without raising the finger tips, by short pushing strokes, centimeter by centimeter, beginning from below and passing upward, until he feels the splash of water under his fingers." Cohnheim requires the patient to inspire deeply and then hold the diaphragm tense, thus pushing the stomach

downward. He advises making strong pressure with the left hand just below the xiphoid process to assist in holding the stomach downward. This expedient undoubtedly depresses the stomach, also it may at the same time result in increasing the tension of the abdominal walls. Owing to individual peculiarities, the relation which the lower border of the stomach bears to the costal arch above and to the umbilicus below, cannot be laid down with great precision. In normal individuals when the stomach is partially filled with water its lower border may be recognized about an inch or two above the umbilicus, but in some individuals it extends only slightly below the costal border. In others in whom the stomach occupies a more perpendicular position, the lower border, which represents the prepyloric portion, may be found to extend as low as the transverse umbilical line, and, in a proportion of cases, in which there is the so-called "fish-hook," or "boot-leg" stomach, the inferior border may reach some distance below the umbilicus.

When there is a thin and relaxed abdominal wall, it is sometimes possible to palpate the pylorus. It is usually found from 2 to 7 cm. above the umbilicus when the stomach is in the normal position. Obrastzow and Cohnheim relate that when palpable the pylorus feels like a cylinder the size of the thumb or forefinger, occupying a horizontal position, running from upper right to lower left and varying in its consistency according to whether it is in a state of relaxation or contraction. Sometimes instead of resembling a cylinder, the pylorus takes the form of a small bunch, the size of the end of the thumb. In addition to this sensation to touch there is sometimes heard a slight sound as of escaping fluid. It is not usually practicable thus to recognize the pylorus, except in very thin people, or when it is unduly resistant. This

over-resistance may result from spasmodic contraction, over-tonicity, or infiltration of the part. When the pylorus is thickened from infiltration or inflammation, the resistance to palpation is more or less continuous, but when it is owing to contraction of the pylorus there is felt upon steady pressure, a relaxation of the part and the resistance disappears and then recurs. It is obvious that the pylorus may be successfully palpated at one time, and yet be undiscoverable at another, depending upon irritability and contractility. Care must be taken not to mistake for the pylorus the so-called "cordecolique" described by Glénard which also occurs as a horizontal resistance located slightly above the umbilicus. and, like the pylorus, is somewhat movable. It has a distinct cord-like feeling, occurs in victims of enteroptosis and by Glénard was supposed to depend upon the empty and relatively contracted transverse colon. Recent X-ray findings show that this interpretation of Glénard cannot be altogether accepted though the presence of the "corde-colique" usually denotes enteroptosis. When the pancreas is indurated it may be palpated through thin abdominal walls. This is especially true of the indurated head of the pancreas. This may be differentiated from the pylorus in that it does not move with respiration nor upon palpation. It lies very deep in the abdomen and through it may be transmitted the impulse of the aorta.

Palpation of the Liver.—To ascertain the topography of the liver it is advisable in the beginning to locate the superior border by percussion. The lower border should be located by palpation. The left hand is placed behind the liver, pressing the ribs forward, while the right hand should rest upon the abdomen, the tips of the fingers pointed upward. The patient is required, with the ab-

domen relaxed, to take a deep inspiration. At the same time, with the left hand, the liver should be pressed still further forward, while the finger tips of the right hand press just sufficiently downward so that the border of the descending liver may be distinguished. It is best to begin palpation with the right hand placed at a point some distance below the margin of the liver, slipping it upwards slightly with each inspiration until the resistance of the hepatic border is recognized. Following this, the patient should be placed upon the left side, the left hand of the examiner pressing the liver forward, while the right hand pushes the abdominal wall inward until the hepatic resistance is noted, and the edge of the organ followed with the fingers as it moves upward and downward with respiration.

The Gall-bladder.—The gall-bladder may protrude from under the liver an inch or two below the costal arch, on a line drawn from the right nipple to the umbilicus. Tenderness of the gall-bladder is usually best recognized by pressing with the tips of one or two fingers at this point. The attempt should be made to discover an enlargement, inequality or irregularity of the liver. Occasionally some doubt arises when there is felt a resisting mass just at the border or below the liver. Then should be considered the possibility of a local enlargement (Riedel's lobe) or tumor of the liver, a dilated gall-bladder, a movable and perhaps enlarged kidney, or tumor of the ascending colon.

The kidney, when movable, may be recognized by making firm pressure forward with the left hand while the right hand is placed firmly on the abdomen, the fingers pointing upward, their tips on a line with the supposed border of the liver. As the patient takes a deep inspiration, the tips of the fingers are pressed forcibly inwards

with the attempt to reach above the kidney and force it downwards, so that it may be further separated from the liver. The same maneuver may be practiced with the patient in the standing position, his hand resting upon a table to steady the body, the weight borne mostly by the left leg, while the right knee is slightly flexed so as to favor relaxation of the right side of the trunk.

Stiller called attention to the fact that in the habitus enteroptoticus there is absence of the cartilage of the tenth rib. The floating tenth rib is usually present in ptosis that develops in young people, and indicates a state of abdominal relaxation and imperfect tonicity. Displacement of viscera may go on progressively for years, and it is sometimes difficult to localize properly the various organs by means of palpation. Under these circumstances it may be advisable to inflate the stomach moderately before palpating and, at another séance to treat the intestines in the same manner. It is the colon, as X-rays studies have taught us, that shows the most unexpected malpositions.

Spleen.—When of normal size and in normal position, the spleen is not palpable. When from enlargement, or ptosis, its lower border extends downwards, it may be recognized sometimes by light palpation, sometimes by deep. The fingers of the left hand, when practicing the latter, should be placed just beneath the left costal border, while the right hand, placed behind, raises the ribs and favors the forward movement of the spleen just at the completion of a deep inspiration. Another method is to place the finger tips of the right hand just under the costal border, requesting the patient to breathe deeply, and at the opportune moment, pressing firmly with the fingers of the left hand over the ninth and tenth ribs,

so that the spleen may be forced downward and thus felt.

Other Organs.—Other organs should be palpated when possible, as for instance the cecum and appendix, the various reaches of the colon, the ovaries, the uterus and the bladder. It is wise to remember the possibility of hernia. One should not forget the small epigastric hernia so often overlooked and so fruitful in producing symptoms.

The Abdominal Aorta.—The abdominal aorta should receive consideration because of the not infrequent presence of aortitis and peri-aortitis which may be recognized by increased sensitiveness, rigidity and motility from side to side. It should be borne in mind that numerous sympathetic ganglia accompany the aorta, and that they are often over-sensitive, either as the result of neurotic conditions or as an expression of abdominal disease and irritation.

Sensitiveness to Pressure.—The degree of tenderness which may be elicited upon palpation and percussion may be of great diagnostic importance. In normal persons the sensitiveness is greater over certain sympathetic points; conspicuous among these is the celiac plexus and the mesenteric ganglia, the latter located on both sides 5 or 6 cm. from the umbilious and slightly below it. (See page 663.) There are several points of less importance in the abdomen where sensation is keener than in the surrounding regions. In neurotic patients, the sensitiveness of the sympathetic ganglia is heightened, sometimes to a marked degree. In hysterical patients, there is sometimes remarkable superficial tenderness which is out of proportion to that of the deeper structures, although the sensation in the sympathetic ganglia may at the same time be greatly exaggerated. There are certain comparatively insensitive individuals who do not resent even deep pressure over any part of the abdomen; in others, however, though in apparently good health, the sympathetics are always keenly sensitive to pressure.

Peptic Ulcer.—The abdominal surface is conspicuously tender upon pressure at a point immediately to the right of the median line and about midway between the ensiform and umbilicus, slightly nearer the former. It is not necessary that the ulcer should be located beneath this point, for the sensitiveness, like the pain of ulcer, seems to be referred. This is not the case with carcinoma of the stomach or perigastritis, in both of which the tenderness corresponds with the location of the structure affected. The degree of pressure necessary to produce pain varies in different affections but in ulcer slight pressure often causes pain.

Instruments for measuring the degree of pressure necessary to induce pain have been invented by Boas, Roux and Shiling. The "algesimeter" of Boas consists of a cylinder inclosing a spiral spring, the compression of which is measured by a scale, indicating the degree of (See illustration, page 652, Fig. 57.) pressure exerted. It has been found that in normal persons, pressure to the amount of 5 to 10 kg. is tolerated. In ulcer 0.5 kg. to 3 kg. produces pain. In carcinoma from 2 kg. to 4 kg. reaches the point of toleration, whereas in nervous cases the point of tolerance does not fall below 4 kg. to 5 kg. The apparatus appears to have value not only in diagnosis. but to indicate the progress of the case. It is distinctly useful to have the means of recording the actual degrees of tenderness. Though not sufficiently reliable unaided by other means for diagnosis, this method has obvious advantages in enabling us to register

quantitatively the sensitiveness, its increase or its subsidence.

#### **PERCUSSION**

The position of the outline of the stomach may be determined by percussion provided the stomach is moderately filled. When empty and in its usual position, it recedes for the most part behind the liver and the left costal arch. Although in case of gastroptosis, the stomach is so placed that it may respond to percussion, it is nevertheless difficult to recognize its outline until the organ is somewhat distended. Percussion should be made from the thorax progressively downward, along the left anterior axillary line, across the ribs and below the costal border. The same plan should be followed along the left parasternal line, the median line and in some cases the right parasternal line. In the horizontal position special tympany is evidenced by light percussion, and the lower border of the stomach may be recognized for the reason that when the outline is passed there is a slight change in the quality, intensity and pitch of the sound.

When the patient is standing, the heavier contents of the stomach gravitates to the lower portion of the organ, the gas occupying the fundus. Under these circumstances percussion made over the stomach elicits dullness except along the axilla, corresponding to the fundus, where there is tympanic resonance. When the patient lies upon the back with the shoulders lower than the hips, a greater amount of gastric tympany may be elicited by percussion over the epigastric region. In case of gastric atony, especially when associated with ptosis, it is with difficulty that the area of the stomach can be determined by percussion. The method is useful, when

the stomach walls are in a tonic condition, in determining the degree of descent of the stomach when the upright position is assumed.

Information concerning the location of the colon may be obtained by percussion, yet here it is less reliable than with the stomach. Percussion is of importance in the study of the general state of the abdomen. Excessive tympany and tension in the mid-abdomen indicate gaseous distension of the small intestines. Marked tympany in the flanks indicates a like condition of the ascending and descending colon respectively, whereas dullness at the flanks suggests the presence of fluid, which may be confirmed through finding shifting dullness. It is therefore advisable to practice percussion with the patient lying first in the prone and then in the lateral posture. Percussion is often of assistance in outlining the lower border of the liver. One should begin far below the organ, make forcible percussion and proceed upwards until hepatic dullness is obtained. Light percussion fails to bring out dullness at the lower border of the liver as the intestines often override the liver at this point.

When there is uncertainty as to the position of the stomach, this may be dispelled by the administration of an effervescing mixture thus inflating the organ before percussion. Four gm. of tartaric acid should be dissolved in half a glass of water and 5 gm. of bicarbonate of soda in an equal amount of water. The patient should be in the lying position, and should rise sufficiently to drink, taking one solution immediately after the other, and resuming position at once. He should be instructed not to bring up the gas. It is advisable to give but half the solution at a time so as to avoid over-distension. This method of distending the stomach is more convenient and satisfactory than that of pumping in air through

a tube. In the event of distress following the introduction of the effervescing mixture, it may be relieved by having the patient sit up, striking the epigastrium sharply with the flat of the hand, and asking the patient to make eructation. Should this fail, the embarrassment is at once relieved by passing the stomach tube. Pain is rarely experienced, except when there is present ulcer, gastritis, or perigastritis. When ulcer is suspected, distension of the stomach should be omitted.

# AUSCULTATION

The deglutition sounds are best obtained by placing the end of the stethoscope at a point beneath the costal border near the ensiform or over the eighth rib in the axillary line. These sounds are produced by the passage of water through the esophagus into the stomach. Meltzer speaks of primary and secondary sounds; the first, produced by the passage of fluid through the lower pharynx, is of a squirting character; and the second, produced by the discharge of fluid through the cardia is bubbling and metallic in character. Five or ten minutes usually elapse between the two sounds. In stenosis of the esophagus, the second sound is delayed. It usually is heard within ten or twelve seconds. Its marked delay or its complete absence is indicative of obstruction usually at the cardia. Revidtzev asserts that in case of obstruction at the cardia, the second sound may be made to recur one or more times by requiring the patient to swallow air. The theory is based upon the belief that with cardiac obstruction there is usually a certain degree of esophageal dilatation, in which case it is unlikely that all the fluid would pass with the first act of deglutition. Repeated acts of swallowing force down the remaining fluid and, with each escape of fluid into the stomach, the sound is repeated. Auscultation over the stomach is of little value for, under pathological conditions when unnatural sounds are produced, they are usually audible without the aid of the stethoscope.

However, stethoscopy associated with friction is sometimes of assistance. The end of the stethoscope should bear lightly over the stomach; then with the finger nail or a spatula the surface is gently scratched; the sound produced by this friction over the stomach is sufficiently distinctive to enable one to outline the organ. Auscultatory percussion over the stomach is also useful in making out the borders, direct percussion in the form of light tapping with the finger being employed.

## CHAPTER III

#### THE GASTRIC CONTENTS

In the preparation of this chapter the aim has been towards clearness and brevity and not to describe numerous methods of examination which, while sometimes necessary, are scarcely required in ordinary work. I am indebted to Dr. Karl F. Eschelman for a concise statement of methods employed in my own laboratory. To these are added a description of a few tests which other workers regard of especial importance.

## GASTRIC JUICE

The gastric juice, chemically is made up of free hydrochloric acid, water, ferments, zymogens, mineral salts and mucus. The hydrochloric acid is secreted by the parietal cells, the proenzymes or zymogens of pepsin and rennin by the chief cells of the peptic and pyloric glands, the mucus by the goblet cells of the gastric mucosa. The zymogens are transformed into ferments by the action of the free hydrochloric acid.

The fasting stomach normally should be empty, but from 30 to 50 c.c. of an acid fluid may be obtained. Anything above this indicates hypersecretion, although there is considerable difference of opinion on this point (Boas vs. Riegel).

The examination of the normal fluid from the fasting stomach shows the following: straw-colored, thin, free hydrochloric acid present in very small amounts; no biuret reaction; no rennin; no lactic acid.

## TEST MEALS

Ewald Test Meal.—This consists of 90 gm. of white or black bread; 300 c.c. of water. The bread should be stale (2 to 3 days old). It is to be thoroughly masticated. At the end of one hour the gastric contents should be withdrawn. Normally 30-80 c.c. is obtained; anything above 200-300 c.c. is abnormal and may indicate one of several conditions, i. e. hypersecretion, lack of motility or pyloric obstruction.

Riegel Test Meal.—This consists of 400 c.c. of beef soup, 100-200 gm. of beefsteak, 150 gm. of mashed potato. The beefsteak must be finely chopped. This test meal is withdrawn after three to four hours.

Germain Sée Test Meal.—This consists of 90 gm. of stale bread (2 to 3 days old), 50 gm. of finely scraped beefsteak, 10 gm. of butter, part of which is spread on the bread and part used in preparing the meal; and 300 c.c. of water.

## GASTRIC CONTENTS

Color.—This depends partly on the test meal used. With the Ewald test meal, the contents normally are of a whitish color. Contents may assume varying shades, from a straw color to a light yellow. At times they are greenish in color, and may even be a grass green. The green color is, as a rule, not due to the presence of bile as popularly supposed, but is probably due to the action of chromogenic bacteria. This has been proven by the fact that such contents will not react to any of the tests for bile or bile salts.

Odor.—Normal freshly withdrawn contents are odorless or have only a slightly sour odor. In contents from cases of stagnation, one may detect the characteristic odor of some one of the organic acids (butyric, acetic) if present, or the odor of yeast fermentation.

Consistency.—This varies from that of a thin fluid to that of a thick gruel, or it may even be that of a viscid mass if much mucus be present. The contents on standing usually will separate into layers. Mucus if present will rise to the top. Under this is a layer of thin turbid fluid. At the bottom will settle the residue of the more solid constituents of the test meal, i. e. bread, meat, etc.

Note the amount of digestion that has taken place in the test meal. The food may be merely macerated and broken up and not digested, as in achylia gastrica, or the contents may consist of a thin turbid fluid containing little or none of the solid constituents of the test meal, as in cases of hypersecretion.

Look for food other than that given in the test meal, the presence of which indicates disturbances of motion, atony, pylorospasm or pyloric obstruction.

#### CHEMISTRY OF THE GASTRIC CONTENTS

#### Actos

Acidity is due to free hydrochloric acid, combined hydrochloric acid, organic acids if present, and the acid salts. Usually the contents will react acid to litmus paper even in those cases in which free hydrochloric acid is absent. Occasionally the reaction may be neutral, alkaline or even amphoteric, as in some cases of gastritis.

Free Hydrochloric Acid.—The most common methods in use for the determination of free hydrochloric acid are those in which color reactions or indicators are used.

QUALITATIVE TESTS FOR FREE HCl.—Günzburg's Solution (phloroglucinvanillin). This is a very reliable test for free HCl. The solution is prepared by adding 2 gm. of phloroglucin and 1 gm. of vanillin to 30 c.c. alcohol. This

should be kept in a tightly stoppered blue glass bottle in a dark place.

Technique.—Place two drops of the phloroglucinvanillin solution in a porcelain capsule with two or three drops of filtered contents. Warm the capsule gently over a flame, not allowing it to become too hot.

When the contents are evaporated almost to dryness, if free hydrochloric acid be present, a rose-colored ring or series of rings will develop at the margin of the solution. The color will vary in intensity depending on the amount of free hydrochloric acid present. This is a valuable test in that inorganic acids only, give the reaction.

Dimethylamidoazobenzole.—The dimethylamidoazobenzole test is a simple qualitative test that is generally employed.

Technique.—Two or three drops of dimethylamidoazobenzole are added to a few c.c. of filtered contents. If free hydrochloric acid be present, a red color appears varying in intensity, in proportion to the amount of free hydrochloric acid present. This indicator also reacts to organic acids and acid phosphates in as high concentrations as might be found in the stomach. Its sensitiveness renders it less accurate for the estimation of free HCl than is Günzburg's reagent.

Resorcin Solution.—This is prepared by making a solution containing resorcin (C. P.) 5 gm.; cane sugar, 3 gm.; 94 per cent alcohol, 100 c.c. This solution is about as sensitive as phloroglucinvanillin and is much more stable.

Technique.—To 5 to 10 drops of filtered contents, add 2 to 5 drops of the resorcin solution, place in a porcelain capsule and warm gently over a flame. Evaporate to dryness. A rose- or vermilion-red mirror will appear if free hydrochloric acid be present. This color mirror fades on cooling.



The presence of organic acids, acid salts, peptones and albumoses does not interfere with the reaction.

QUANTITATIVE ESTIMATION OF HYDBOCHLOBIC ACID.—Sahli Method.—Titrate 10 c.c. of filtered contents with nNaOH until no reaction for free hydrochloric results. Add the nNaOH drop by drop. At intervals remove a drop of the contents on a glass rod and test it for the presence of free hydrochloric acid, using Günzburg's reagent. Since a small amount of the contents is lost each time the test is made, the end result should be confirmed by taking a new portion and again titrating. Only the free HCl enters into the reaction, the combined HCl, acid salts and organic acids not occurring in such concentrations as to interfere with the test.

Töpfer's Method.—To 10 c.c. of filtered contents add one drop of 0.5 per cent dimethylamidoazobenzole. Titrate with  $\frac{n}{10}$ NaOH until the red color disappears, and is replaced by a distinct canary yellow color.

The degree of acidity is considered to be represented by the number of c.c. of  $\frac{n}{10}$ NaOH required to neutralize 100 c.c. of contents. Töpfer's method is the most simple for the general practitioner. It includes besides the estimation of free HCl, as above given, that of total acidity, loosely combined HCl, organic acids and acid salts. The complete test is given below:

Total Acidity, Free HCl, Loosely Combined HCl, Organic Acids and Acid Salts.—Töpfer's Method.—The following solutions are required: nNaOH; phenolphthalein, 1 per cent alcoholic solution; alizarin (neutral sodium salt), 1 per cent aqueous solution; dimethylamidoazobenzol, 0.5 per cent alcoholic solution.

(a) The total acidity of a given amount of contents (i. e. the acidity due to free hydrochloric acid, loosely combined hydrochloric acid, acid salts and any organic acids that may be present) is estimated using phenolphthalein as an indicator.

- (b) The acidity due to free acids and acid salts is estimated using the same amount of contents with alizarin (alizarin monosulphate of sodium) as an indicator.
- (c) The free hydrochloric acid is estimated using the same amount of contents, with dimethylamidoazobenzol as an indicator.

Technique.—Measure 10 c.c. of filtered contents into each of three porcelain capsules. To No. 1. add 2 drops of phenolphthalein, titrate with  $\frac{n}{10}$ NaOH until a deep red color appears and is permanent and does not turn deeper on the addition of a further drop of  $\frac{n}{10}$ NaOH, this point being the end reaction.

To No. 2. add 2 to 3 drops of the alizarin solution and titrate with  $\frac{n}{10}$ NaOH until a pure violet color is obtained, which shows neutralization of all the acidity of the filtrate except that due to combined HCl. Estimating the alkali required for 100 c.c. of contents and subtracting from the total acidity, we have the acidity due to combined HCl.

To No. 3. add 2 to 3 drops of dimethylamidoazobenzol and titrate with  $\frac{n}{10}$ NaOH until the last traces of red in the solution disappear leaving a canary yellow color.

The results are calculated as follows:

Example.—Ten c.c. of contents, using phenolphthalein as an indicator, required 7.2 c.c. of ANOH to produce the end reaction. Also ten c.c. titrated in a like manner, using alizarin as an indicator, required 6.2 c.c. to produce the end reaction. Also ten c.c. titrated as the above, using dimethylamidoazobenzol as an indicator, required 5.2 c.c. to produce the end reaction.

Therefore the total acidity would be  $7.2 \times 10$  or 72.0 degrees.

The loosely combined hydrochloric acid would be 10 degrees ( $6.2 \times 10$  or 62.0 degrees; this subtracted from the total acidity gives the amount of loosely combined hydrochloric acid, i. e. 72.0 - 62.0 or 10 degrees).

The free hydrochloric acid would be  $5.2 \times 10$  or 52.0 degrees.

The organic acids and acid salts would be the total acidity minus the free hydrochloric acid and the loosely combined hydrochloric acid, i e. 72.0 - (52.0 + 10) = 10.0. degrees.

The estimations made above are represented in "degrees," that is, in terms of the number of c.c. of decinormal sodium hydrate required to neutralize 100 c.c. of contents. They may be figured in percentage direct as to HCl by taking into account the fact that one c.c. decinormal sodium hydrate neutralizes .0036 gm. HCl. Thus, in the example given, 52 c.c. of the alkali were required for 100 c.c. of contents in testing for free HCl. Fifty-two c.c. must have neutralized  $52 \times .003637$  or .189 gm. HCl per 100 c.c. of contents.

Lactic Acid.—The presence of lactic acid is of value only when a test meal has been given which is absolutely free from lactic acid. The method of Boas may be used. It consists in washing the stomach clear at night, then giving in the morning oatmeal gruel seasoned with a little salt. Avoid meats in a test meal when lactic acid is to be determined, as sarcolactic acid is present as a preformed acid in meats.

Tests.—Uffelman's test.—Uffelman's reagent is made by mixing 10 c.c. of 4 per cent carbolic acid solution, 20 c.c. of distilled water, and 1 drop of the officinal liquor ferri chloridi. The resulting solution is a clear amethyst blue. To a few c.c. of the reagent add 5 to 10 drops of filtered contents. Lactic acid, if present, will turn the blue solution to a yellow or yellowish green.

Kelling's method.—Take 5 to 10 c.c. of filtered contents, dilute with 10 to 20 c.c. of distilled water, add 2 drops of a 5 per cent aqueous solution of ferric chlorid. If lactic acid be present, a greenish yellow color will be seen when the test tube is held against the light.

This test is more reliable than that of Uffelman, and is positive only to lactic acid.

Butyric Acid.—The presence of this organic acid in the gastric contents may be recognized usually by its characteristic odor, that of rancid butter; or more accurately by the following test: Extract 10 c.c. of filtered contents with 50 c.c. of ether, evaporate the ether, dissolve the residue in a small amount of water, add a small amount of calcium chlorid and the butyric acid will separate in the form of oil droplets which will have the characteristic pungent odor of butyric acid.

Acetic Acid.—This organic acid may also be recognized by its odor, or by the following test: Extract 10 c.c. of filtered contents with 50 c.c. of ether, evaporate the ether, dissolve the residue in a little water, neutralize accurately with  $\frac{n}{10}$ NaOH, using phenolphthalein as an indicator. To this solution add a few drops of a very dilute ferric chlorid solution. If acetic acid be present a dark red color will result.

## FERMENTS AND ZYMOGENS

Pepsin.—The precursor of pepsin is the zymogen pepsinogen which is secreted by the chief cells of the glands in the fundus of the stomach. In the quiescent stomach only inactive pepsinogen is found. During the process of digestion pepsin is found. Pepsin is active only in

PEPSIN 55

acid solutions. Weak alkaline solutions will inhibit or destroy its action.

BIURET TEST FOR PEPTONES.—A small amount of filtered contents is rendered strongly alkaline by the addition of a small amount of potassium hydroxid. Then a few drops of a 1—1000 copper sulphate solution are added. A rose-red color indicates the presence of peptones, hence the presence of pepsin may be inferred. Although this test has been generally used its value has been over-estimated as the same color reaction is given by other proteids than peptones. The dividing line between the reactions of the various proteids is difficult to judge with an untrained eye. The biuret reaction may be present in any stomach contents regardless of the presence of pepsin.

QUALITATIVE TEST FOR PEPSIN AND PEPSINOGEN.—If free hydrochloric acid be present, 10 c.c of filtered contents are placed in a test tube and several discs of coagulated egg albumin are added. (These are prepared as follows: Boil an egg five minutes, then cut the coagulated white of the egg into discs 5 mm. in diameter, by 1 mm. thick. Keep in glycerine until wanted. Before using wash thoroughly in water.) Place the test tube in the incubator at 37.5° C. and leave for one-half to four hours. The first sign of digestion taking place is the rounding of the sharp edges of the discs.

If free hydrochloric acid be absent, the contents are rendered acid with a few drops of hydrochloric acid and the above technique is carried out.

QUANTITATIVE ESTIMATION OF PEPSIN.—Method of Hammerschlag.—Hammerschlag's solution consists of a one per cent solution of albumin containing 4 per mille of hydrochloric acid. This is most conveniently made in the following manner (Cohnheim):

Sixteen c.c. of concentrated hydrochloric acid is mixed with 1,000 c.c. of tap water. This solution is slowly stirred into 140 c.c. of fresh egg albumin. The resulting mixture is then filtered through a piece of fine mesh cheesecloth arranged in a funnel.

The solution should be made up freshly every two to three weeks as the amount of egg albumin decreases through decomposition.

Technique.—Ten c.c. of Hammerschlag's solution is placed in each of two test tubes. These are marked respectively P (patient) and W (water). Five c.c. of filtered gastric juice is added to P, and 5 c.c. of distilled water is added to W. The test tubes are then placed in a beaker containing water at a temperature of 38° to 40° C. and allowed to remain there until they reach the temperature of the surrounding water. They are then taken out and placed in the incubator for one hour at a temperature of 38° to 40° C. At the end of this time they are taken from the incubator and placed in a beaker of cold water to inhibit further peptic activity. Two Esbach tubes are marked to correspond to the test tubes Esbach tube P is filled to the U mark with the contents of the test tube marked P. Esbach tube W is filled to the U mark with the contents of the test tube marked W. Both Esbach tubes are now filled up to the R mark with Tsuchiya's reagent (phosphotungstic acid, 1.5 gm.; concentrated hydrochloric acid 5 c.c.; ethyl alcohol, q.s. ad. 100 c.c.). This reagent gives, according to Goodman and Stern more accurate results than Esbach's original solu-The tubes are closed with rubber corks and shaken well, then allowed to stand for twenty-four hours at room temperature. At the expiration of this time the height of the albumin column in each tube should be read.

Example.—If the albumin column in the Esbach tube

P which contained the gastric juice is at 1 per mille, and in the tube W containing the water at 6 per mille, then there would be 5 parts out of the 6 parts of albumin peptonized, that is five-sixths digested or 83½ per cent.

Hammerschlag states that normal pepsin digestion is from 70 to 80 per cent; in hyperchlorhydria, 90 per cent; while in cases of hypochlorhydria, as low as 10 per cent, or even lower in an acidity.

Method of Mett as Modified by Elsner.—Capillary tubes of 2 mm. diameter (internal cross section) and 20 to 25 cm. in length are filled with fresh egg albumin. These are boiled until the albumin is coagulated. They are then removed and cut with a file into pieces 3 cm. long. Several of these pieces are placed in a beaker with 5 to 10 c.c. of the gastric juice whose digestive activity is to be estimated. The beaker and tubes are placed in an incubator and allowed to remain for twenty-four hours and then removed. The digested portion of the albumin column at both ends of the tube is measured by means of a millimeter measure and a magnifying glass. Normally this ranges from 5 to 12 mm.

The estimation of the relative amount of pepsin is made according to the law of pepsin activity (Schütz), the amount of formed peptone being calculated from the length in millimeters of the albumin column digested in the Mett's tubes.

The pepsin varies as the square of this length; that is, a digested portion of 2 mm. represents 4 parts of pepsin; 5 mm. represents 25 parts, etc.

Method of Gross.—One gm. of casein (caseinum purissium-Grubler, prepared after the method of Hammarsten) is added to 16 c.c. of 25 per cent hydrochloric acid (specific gravity of 1.124) made up to one liter with water and dissolved on a water bath.

Ten c.c. of the casein solution, previously warmed to a temperature of 39° to 40° C. are placed in each of a series of test tubes. To these are added graded amounts of the gastric juice to be examined, the dilution depending on the peptic activity of the gastric juice. (Gross apparently dilutes the gastric juice and uses amounts of this dilution equal to 0.01, 0.02, 0.03, 0.04, 0.05, etc., of the gastric juice.)

The tubes are then placed in an incubator or water bath at 39° to 40° C., for 15 minutes, at the expiration of which they are taken out. Then a few drops of a concentrated solution of sodium acetate are added to each tube. The presence of casein remaining undigested is shown by a clouding up of the contents of the tube.

He assumes the normal of pepsin to be the amount contained in a gastric juice, exactly 1 c.c. of which is sufficient to digest all the casein in 10 c.c. of the solution (which contains 0.01 gm. of casein) in just 15 minutes.

Example.—If it takes 0.125 c.c. of the gastric juice to digest all of the casein in 10 c.c. of the solution in 15 minutes, then the peptic activity of the gastric juice is 8 units. Similarly, 0.05 c.c. of gastric juice represents 20 units; 0.025, represents 40 units; 0.0125, represents 80 units.

Gross gives 30 to 50 as a standard for the peptic units of normal gastric juice.

Rennin and Its Zymogen.—The presence of rennin may be determined as follows: Take 10 c.c. of milk, add five drops of filtered contents and incubate 10 to 20 minutes at 37.5° C.. If the milk coagulates, it may be presumed that rennin is present. If a more accurate determination be wanted, the following technique may be followed. Put 10 c.c. of milk, raw or, better, boiled, in a test tube, add 5 c.c. of contents that have been accurately neutral-

ized with no NaOH. Place this in the incubator for 15 to 30 minutes at 37.5° C. A flocculent coagulum is formed if rennin be present.

ZYMOGEN.—Take 10 c.c. of milk, add 5 c.c. of filtered contents that have been made feebly alkaline with  $\frac{n}{10}$  NaOH, then add 2 c.c. of a 1 per cent calcium chlorid solution. Place in a test tube and put in the incubator at 37.5° C. for 10 to 15 minutes. At the end of that time if zymogen be present, a firm coagulum of casein will have formed.

QUANTITATIVE ESTIMATION OF RENNIN AND ITS ZYMOGEN.

—Method of Boas.—This method is based on the fact that on gradually diluting a specimen of gastric contents a point is reached at which no further rennin action can be obtained.

The filtered gastric contents are exactly neutralized with a decinormal solution of sodium hydrate. A series of test tubes are prepared containing from 5 to 10 c.c. of gastric contents in the following dilutions,  $\frac{1}{10}$ ,  $\frac{1}{20}$ ,  $\frac{1}{30}$ ,  $\frac{1}{40}$ , etc. To each test tube neutral milk is added in amount equal to the amount of gastric contents used. The tubes are then placed in an incubator at a temperature of 37° to 40° C., and observed from time to time. The lowest degree of dilution at which coagulation takes place is then noted. This, according to Boas, occurs in normal contents at a dilution varying between  $\frac{1}{30}$  and  $\frac{1}{40}$ .

Rennet Zymogen.—The filtered gastric contents are rendered weakly alkaline with a solution of potassium hydrate, and the dilutions and determinations are made the same as for rennin. Coagulation occurs in normal contents at a dilution of  $\frac{1}{100}$  to  $\frac{1}{100}$ .

Starch digestion may be tested for by the use of a very dilute Lugol's solution. A few drops of filtered contents

are added to a very dilute Lugol's solution. Soluble starch or amidulin will give a blue color, erythrodextrin will give a violet to a mahogany brown, achroödextrin may be assumed to be present if there is no change in color.

# Mucus

Normally a small amount of mucus is present in all contents. It may be recognized macroscopically by pouring the contents from one vessel to another. If much mucus be present the contents will be viscid and stringy or ropy. Swallowed mucus coming from the upper air passages may easily be differentiated from that secreted by the stomach, in that it usually floats on the surface of the contents because of the air it contains, and in that it is not intimately mixed with the contents but occurs in lumps and masses. The mucus secreted by the stomach is intimately mixed with the food and usually occurs in small flakes. It may be present in such large amounts that the contents forms a sticky, glairy mass.

#### BLOOD

Blood may be present, macroscopically, microscopically, or in such form that it may only be detected by some one of the numerous tests for hemoglobin.

Guiaiacum Test for Occult Blood.—Five c.c. of gastric contents are mixed with 3 c.c. of glacial acetic acid, and extracted with 5 c.c. of ether by shaking gently. The mixture is allowed to settle and the ethereal extract is then decanted. One c.c. of a fresh tineture of guiaiac solution is placed in a porcelain capsule and 2 to 3 c.c. of the ethereal extract is added and well mixed. Then 1 c.c. of fresh hydrogen peroxid is added. If blood be present a bluish violet color develops.

Benzidin Test (Method of Schlesinger and Holst).—Put 5 c.c. of gastric contents in a test tube, heat over a flame bringing the contents to a boil, then allow contents to cool. Make a saturated solution of benzidin (Merck) by dissolving a small amount in 2 to 3 c.c. of glacial acetic acid in a clean test tube. Place 1 c.c. of the fresh benzidin solution and 2 to 3 c.c. of a fresh commercial 3 per cent solution of hydrogen peroxid in a clean porcelain dish. No change in color to a green or blue should occur in this mixture. Next add a few drops of the previously prepared gastric contents. If blood be present a beautiful green, bluish green to blue color will appear. The depth of color depends on the amount of blood present in the contents. Very slight changes in color should be disregarded.

This is a very sensitive test, and when negative excludes the presence of even minute quantities of blood.

#### CARCINOMA

Salomon's Test.—The fasting stomach is carefully washed out in the evening preceding the test. The next morning the stomach is carefully irrigated several times with 400 c.c. of a normal saline solution, this being used over and over.

The albumin and the nitrogen in the wash water are then carefully determined. The presence of over 0.5 gm. per liter of albumin (using Esbach's tube with Tsuchiya's reagent) is supposed to be indicative of carcinoma. In all other gastric conditions the nitrogen equals from 0.0 to 0.0016 gm. (as determined by Kjeldahl's method), but in carcinoma the nitrogen equals from 0.0020 to 0.0070 gm. per 100 c.c. Carcinoma is probable when the nitrogen is more than 0.0020 gm.

Tryptophan Test (Neubauer and Fischer).—This test is

based on the theory that carcinoma of the stomach will secrete a ferment which exerts a peptid-splitting action.

After the test meal is withdrawn it is tested for the presence of blood and bile. If these are absent, the test for the ferment is made.

To 10 c.c. of filtered contents, a small amount of glycyltryptophan is added; cover with a layer of toluol and place in the incubator for 24 hours. At the end of that time it is taken out. Pipet off 2 to 3 c.c. of the liquid under the toluol and place it in a test tube, adding a few drops of 3 per cent acetic acid. Then carefully add bromin vapor from a bottle containing bromin by allowing the vapor to fall on the top of the contents of the test tube. Shake the test tube and if the contents of the test tube assume a rose color, free tryptophan is present. If the color does not appear, repeat the procedure carefully until an excess of bromin is present as indicated by the yellow color of the contents of the test tube. If the color still does not turn to a rose color, the test is negative.

# MICROSCOPICAL EXAMINATION

Oppler-Boas Bacilli.—These are found in 70 to 80 per cent of all cases of carcinoma, seldom in other conditions. These bacilli are 3 to 10 microns long by 1 micron in width, may form chains, stain with the ordinary anilin dyes, are gram-positive. They stain brown with iodin, which differentiates them from Leptothrix from the mouth which stain blue with iodin, although Fricker states that the Oppler-Boas bacilli in a number of instances show an undoubted starch reaction, in other words these bacilli sometimes show blue granules on being treated with Lugol's solution. (See pages 442-446. Figs. 35-41.)

The Oppler-Boas bacillus gives rise to lactic acid. It is found in the stomach only when there exists stagnation of contents and low secretion of, or absence of, hydrochloric acid. It has been found in the oral cavity, the esophagus and the colon; but only when there are accumulations which are not actively passed along by the motor function of the part. Thus it may occur in sacculations, diverticuli and dilations. The advantage of recognizing the Oppler-Boas bacillus in the stomach contents lies in the fact that when the organism is found it is equivalent in the point of diagnosis to the finding of lactic acid, and the bacillus may be discovered in a quantity of gastric contents too small to permit of a reliable chemical test for lactic acid.

Sarcinae.—These are found in cases of gastric dilatation, rarely in normal cases. They are associated with marked fermentation and high acidity. They consist of cocci arranged in bale-like packets. They are not pathologic but are indicative of stagnation.

Yeast Cells.—They are found in all contents that have had bread as the basis of the test meal. Unless they are present in large numbers they are to be disregarded. They occur in cases of marked fermentation and high acidity, associated usually with sarcinae.

Epithelium.—In cases of gastritis, epithelium and small shreds of the gastric mucosa may be found in the contents. Larger shreds and fragments are found in cases of the so-called Einhorn's disease and carcinoma of the stomach. (See Figs. 8, 9, 10, 11, page 64.)

# TESTS FOR MOTOR ACTIVITY OF THE STOMACH

Moritz found that if 500 c.c. of distilled water be taken in a fasting stomach, it will be found to have passed into the intestine from the normal stomach within 45 minutes.



FIG. 8.—BENIGN STAGNATION

- 1. Sarcinae ventriculi.
- 2. Yeast cells.
- 3. Starch granules.
- Fat droplets.
   Plant cells.
- 6. Fat crystals.
- 7. Mucus
- 8. Pus cell.
- 9. Epithelial cells.

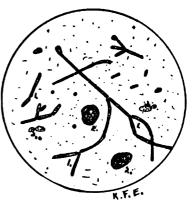


Fig. 9.—Pencillum GLAUCUM, FOUND IN GASTRIC CONTENTS

- 1. Pencillum glaucum.
- Starch cells.
   Yeasts.

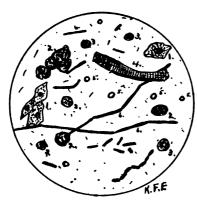


FIG. 10.—GASTRIC CONTENTS FROM A CASE OF CARCINOMA

- 1. Epithelial cells.
- 2. Starch cells.
- 3. Pus cells.
- 4. Muscle fiber.
- 5. Red blood cells.
- 6. Long bacilli. (Faden bacilli.)
- 7. Strepto bacilli.
- 8. Short bacilli.



11.—SEDIMENTED GASTRIC CONTENTS FROM A FASTING STOMACH

- 1. Yeast cells.
- 2. Epithelial cells.
  3. Mucus spirals.
- 4. Free nuclei.
- 5. Mucus shred.
- 6. Staphylococci.

A more precise method, recommended by Boas <sup>1</sup> is a modification of this. When fasting, the patient drinks 400 c.c. of water including 20 drops of a strong aqueous solution of chlorophyl, a substance not absorbed by the gastro-intestinal mucosa.

With normal motility about 50 c.c. remains after half an hour. The precise amount remaining may be recovered by lavage and estimated by the colorimeter.

For practical purposes the estimation of the residuum of an Ewald test breakfast is sufficient. It is inadequate when doubt exists as to whether or not the stomach has been completely emptied by the stomach tube.

Calculation of the Total Contents of the Stomach at a Given Time for Estimating Motor Activity and Oversecretion.—Actually, all delay in emptying of the stomach is accompanied by hypersecretion of reflex origin. When delay in emptying is but moderate it often happens that the fasting stomach in the morning has emptied itself of all traces of aliment and that the fluid present represents only the result of hypersecretion. In order to be certain of the nature of the motor disturbances of small degree it is necessary to have recourse to precise methods of examination, as these alone enable us to distinguish pure hypersecretion from hypersecretion united with delay in emptying the stomach.

The test most advisable for determining exactly the motor functions of the stomach is one based upon the principle proposed by Mathieu and Remond and usually called the "remnant test."

An hour after taking an Ewald test breakfast as much as possible of the stomach contents is withdrawn through the stomach tube. There is then introduced through the tube a known quantity of distilled water. The funnel is

<sup>1</sup> Deutsch, Med. Woch., 1912, XXXIII.

lowered until a good part of the diluted gastric contents is present therein. The funnel is then raised until the diluted contents have mostly re-entered the stomach; then again the funnel is lowered and again raised, the process being repeated several times until a perfect mixture in the stomach of the diluted contents is obtained. Then there is extracted a specimen of the diluted gastric juice which is received in a special receptacle.

Let x represent the volume of the gastric contents which has not been extracted from the stomach, and to which is added the known quantity of distilled water, which is represented by q.

Let v represent the quantity of pure gastric juice primarily extracted and let a represent the acidity of this liquid and a' the acidity of the liquid by the quantity of water q. The total quantity of acid contained in the liquid left in the stomach remaining always the same whether this liquid be diluted or not, we are able to establish the following equation:

$$ax = a'q + a'x$$

from which

$$x = \frac{a'q}{a - a'}$$

The quantity of contents originally in the stomach may be represented by the formula

$$V = v + \frac{a'q}{a - a'}$$

· V represents the total volume of the gastric contents at the moment of the introduction of the stomach tube.

<sup>&</sup>lt;sup>2</sup> Mathieu et Remond.

## CHAPTER IV

### THE STOMACH TUBE

The modern advance in the knowledge of gastric pathology began with the use of the stomach tube for the withdrawal of stomach contents and the study of the same with relation to digestion. The stomach tube continues to be the most important instrument in the diagnosis and treatment of stomach diseases. Failure to obtain satisfactory results depends mostly upon either an improper tube or lack of expertness in using it.

The stomach tube should not be too small; for an adult it should be from 30 to 35 French scale. It should not be too flexible; it should support its own weight without bending when held in the upright position for the length of twelve inches. It should have the largest caliber possible without being liable to bend sharply at an angle when pressed on. A good deal depends on the quality of the rubber; much depends on the character of the tube. It should have no pumping apparatus connected with it and it should be about 54 inches long.

On introduction the tube is occasionally grasped by the esophagus about 8 or 10 inches from the teeth. If this delay is temporary, it is caused by spasm, and may be accompanied by either cardiospasm or stricture at the cardia.

The return of a portion of the test meal when the tube has passed about 12 or 14 inches, the food being unmixed with gastric juice, is evidence of obstruction of some kind at the cardia and the retention of food in the esophagus, which is probably dilatated.

If in pressing the tube onward it stops at the cardia or, being engaged by a spasmodic contraction, is held fast, we may infer the existence of stricture or spasm at the end of the esophagus.

On passing the tube into the stomach we usually obtain



FIG. 12.—THE STOMACH TUBE DESCRIBED. Exact size reproduced.

the best reflow at a distance of from 21 to 22 inches from the teeth, provided the patient is an adult of ordinary stature, with the stomach in position and of normal size.

When the stomach is high the reflow may be obtained at 18 inches. In a very tall person the tube may have to be passed 24 inches or beyond, even when the stomach is in usual position.

When the tube is passed 20 to 22 inches we may find stomach contents; and on aspirating this and pressing

the tube further on, after a little resistance it apparently gets into a second cavity from which stomach contents of a different character may be removed. This indicates an hour-glass stomach or a spasmodic contraction of the stomach between the middle and last third of the organ; in other words, at the antrum pylori.

When, after passing the tube the usual distance at the proper time after a test meal, we find present an unusually large amount of the meal, it indicates moderate motor insufficiency of the stomach which may depend on atony or on some opposition at the pylorus.

We may find a large quantity of the test meal present undergoing fermentation and having a "musty" odor. It may show a large quantity of bacteria and sarcinae and a full secretion of hydrochloric acid with a few lactic acid bacilli. This condition is indicative of benign obstruction at the pylorus.

If we find stagnating food of foul odor, either with or without free hydrochloric acid, with lactic acid, the Oppler-Boas bacilli and unchanged or occult blood, malignancy and obstruction are suggested.

The presence of too little of the test meal at the usual time, suggests over motor activity.

When we find the stomach empty or find present too little of the test meal and no gastric juice, it indicates excessive motor activity, probably associated with achylia gastrica (frequently present in pernicious anemia).

A conclusion should not be reached by a single examination. On one day there may be found an absence of gastric secretion and on the next an excess of it. On one day the food may be retained beyond the usual time and on another it may have passed onward too quickly. If such is the case we conclude that the patient is subject to

nervous disturbance, perhaps excited by the very practice of lavage.

When we find habitually an acidity above 60, depending on hydrochloric acid, free or combined, the presence of hyperchlorhydria is suggested.

Hyperchlorhydria, however, is not to be determined by a given standard. The secretions of the stomach are seldom stable and do not submit to precise standards. A secretion normal with one person is excessive with another. In other words, some patients suffer from the symptoms of hyperchlorhydria when the secretion is decidedly below the standard. Such patients may be victims of gastric hyperesthesia.

There are cases of practical suspension of the gastric secretory function with symptoms similar to those of hyperchlorhydria. These are probably instances of chronic glandular gastritis with exacerbations accompanied by symptoms of irritation.

Over-acidity with delay in digestion of starch is indicative of hyperchlorhydria.

A high total acidity, depending mostly on free hydrochloric acid, suggests a deficiency in secretion of enzymes.

A high total acidity, depending largely on combined chlorids, suggests an active secretion of the enzymes.

The presence of mucus in excess should always attract attention. A large quantity of glairy, ropy mucus, unmixed with the test meal, is probably esophageal mucus which has entered the stomach through the irritation occasioned by the passing of the tube. It often occurs when the tube has been introduced in a clumsy manner. It is not indicative of gastritis.

Mucus in nummular masses often accompanied by pus and by epithelium from the respiratory tract, has been swallowed and is not of stomach origin. When mucus is found thoroughly intermixed with the test meal, usually not glairy or ropy, it probably comes from the gastric mucosa, and is indicative of catarrhal gastritis.

The presence of occult blood or blood cells having undergone change by digestion is evidence that hemorrhage has taken place in the stomach with active secretion such as occurs in ulcer.

If the blood cells are found intact it speaks for recent hemorrhage or lack of digestive power or both.

The presence of blood with mucus and pus cells, apparently not coming from the respiratory passages, with fragments of tissue, suggests ulceration or degeneration of the gastric mucosa and probably malignancy.

A slow return of the gastric contents through the tube, showing little pressure from below, is indicative of atony and probably of dilatation.

Finding food remaining after washing for a considerable time is evidence of atony and probably dilatation.

Finding that the stomach holds a large quantity of fluid without inconvenience, is indicative of dilatation. However, testing the capacity of the organ in this manner is a practice not to be recommended.

On the other hand, if the stomach contents return through the tube with considerable force, it indicates a strong motor activity or a stomach greatly overdistended with contents, certainly a high pressure. Under such circumstances we should attempt to rule out the element of strong contraction of the abdominal muscles.

A forcible return may occur in dilatation provided there is hypertrophy of the muscle walls as a result of pyloric stenosis.

Violent or spasmodic spurts, coming from the stomach

tube, result from sudden contraction of the abdominal muscles and a suddenly increased intra-abdominal pressure.

When bile is found in the stomach it probably depends on relaxation of the pylorus with upward pressure of the duodenal contents through contraction of the abdominal muscles or reverse peristalsis.

When the proximal end of the tube is held beneath water and numerous bubbles of gas escape through it, it is indicative of an excess of gas in the stomach.

When no gas escapes from the stomach, but the patient complains of continuous eructations, it may be assumed that the gas is not formed in the stomach by fermentation, but that we are dealing with an hysterical condition.

Slow change of albumen, especially of meat fibers, shows that the gastric digestion is retarded.

The odor of fatty acids, or a "musty" or vinous odor, is evidence of fermentation.

Too much importance should not be attributed in stomach examinations to the disturbances in secretion, but great importance should be attributed to evidence of disturbances in motion.

The diagnosis of stomach diseases should not be made solely by examination of the gastric contents, but by the results of this method considered in connection with the symptomatology and with the estimation of all the facts in the case.

Wrong conceptions will arise from basing a diagnosis on stomach examinations alone, or on too infrequent examinations; or on examinations conducted without rules and without thought. The stomach tube should be much more commonly employed as a routine, in general examinations than it is but, as in other physical methods of

examination, the results will depend on the expertness and thoughtfulness of the examiner.

The stomach tube is of the greatest importance in the matter of diagnosis and may be equally so in treatment; however as employed it is often useless and sometimes pernicious. It is at times a therapeutic agent of great efficiency and cannot be replaced by any other measure as in poisoning cases, gastrectasis, before operation in obstruction, in some cases of intractable vomiting, in catarrhal gastritis and, in general, for the relief of an irritable gastric mucosa.

Technique of Using the Stomach Tube.—The patient should sit in an ordinary straight-back chair. Fastened about the patient's shoulders there should be a long rubber apron, reaching to the floor. A towel should be tucked in around the neck. The physician stands at the patient's right side, dips the tube in cold water and proceeds to its introduction. The patient is instructed not to bend the head forward or backward, but to hold it in the natural position. It is best not to excite the patient's apprehension by too many preliminary explanations. He is instructed to protrude the tongue slightly; the tube is then slipped into the fauces, the point directed downward and the patient is requested to swallow. At the same moment, the tube is pushed onward beyond the glottis and the patient commanded to inhale, to direct all his attention upon breathing. Meanwhile the tube is slipped down gradually and, with each attempt at retching or the beginning of strangling, the patient is again commanded quietly but firmly first to swallow then to breathe. Very soon the extremity of the tube enters the stomach descending to the desired point, which is ascertained by the marks along the tube. At the first séance it is well for an assistant to hold the tube a little to one side, so as

to avoid the epiglottis, at the same time steadying the hand against the patient's chin, to avoid unnecessary movement of the tube that is prone to excite vomiting. Meantime the operator compresses the tube between the thumb and finger until he is ready for the expulsion of the stomach contents. The patient is then directed to bend forward from the waist, without dropping the chin, and to bear down as though moving the bowel. The increased intra-abdominal pressure thus produced is sufficient to express pure stomach contents, provided the tube is in the right position, not blocked by a morsel of food and not compressed by spasm at the cardia. When the aspiration is not successful it is inadvisable to introduce water into the tube "to start the flow." This practice dilutes the stomach contents and largely vitiates the result of the examination. Nor is it often advisable to attach an aspirating bulb for the purpose of sucking out the contents. If not successful, the tube should be further introduced, or slightly withdrawn, until the proper level is reached, each time obtaining the cooperation of the patient to increase the intra-abdominal pressure. Sometimes coughing will suffice to start the reflow when bearing down is ineffectual. Should the stomach contents fail to appear, lavage should be practiced and an appointment made for another trial. Success or failure in aspiration depends largely on the manner of the physician and the moral control which he has upon his patient. The stomach contents should be received in a small glass or porcelain basin. When the patient is not over-fatigued or when time is important, lavage now should be practiced. For this, sterile water at the temperature of the body should be at hand to the amount of several gallons. A definite amount should be poured into the tube in a steady stream, never allowing the funnel to

become empty, thus preventing the entrance of air into The returning wash-water should be rethe stomach. ceived in a graduated vessel, so that it may be known that the amount introduced has been fully discharged, and thus distension of the stomach avoided. Additional receptacles should be at hand so that any particular specimen, possibly containing stomach contents of different appearance, possibly mucus, blood or tissue, may be preserved separately for examination. The stomach should be washed until the water returns clear. It is sometimes necessary to wash out the stomach when the patient is in bed. When so, he should lie on his left side with the shoulders slightly elevated and thereafter the procedure corresponds to that just described.

COUNTER-INDICATIONS.—The stomach tube should not be used in case of acute, bleeding ulcer, when cerebral hemorrhage or hemoptysis are threatened, when there is aneurysm, or in threatened abortion. It is best not to practice lavage during the menstrual period.

Gastro-diaphane.—This ingenious instrument, introduced by Dr. Max Einhorn, although largely superseded by the radioscope, should not be altogether disregarded. The apparatus consists of a rubber tube, provided at its distal extremity with a small electric lamp, which is connected by wires passing through the tube with a suitable dry cell or storage battery. The examination should be conducted in a darkened room. The patient should drink about two glasses of water, then the instrument is introduced like a stomach tube. With the abdomen exposed and the current turned on, the illumination produced by the electric light appears through the abdominal wall as a pale, rose colored area, changing in location as the tube is moved. To prevent over-heating the surface of the stomach, the current should be occasionally interrupted

and the position of the instrument slightly changed. The position of the stomach and suitably located tumors and deformities thus may be determined.

### CHAPTER V

### THE STRING TEST

In clinics where it has been thoroughly and conscientiously tried, the "string test" of Dr. Max Einhorn has won a reputation as a comparatively reliable method in the diagnosis of peptic ulcer. It also assists in the diagnosis of other diseases.

The apparatus consists of a round, braided surgeon's silk. No. 5, of sufficient length, at the end of which is a small hollow bulb or bucket about 5 mm. in diameter. The patient is required to swallow this bulb, which may be inclosed in a gelatine capsule; the proximal end of the thread is fastened to the ear or at the neck of the patient. The point where it passes the incisor teeth is at a known distance from the bucket, the thread being marked for the convenient estimation of the length that should be swallowed, about 70 to 80 cm. This silk is kept in place ordinarily for twelve hours, the patient fasting; or the stomach may be thoroughly emptied by lavage in the evening, and the thread introduced and worn until morning. The bucket end readily finds its way past the pylorus and is withdrawn without any or with but slight, inconvenience. When the pylorus is unusually irritable, the patient is made aware of the onward passage of the bucket through the pylorus by the occurrence for a few moments of a sense of constriction or a slight, peculiar, pricking sensation felt to the right of the median line a little below the costal border. When the pylorus is irritable, the withdrawal of the thread is delayed as the bucket passes from the duodenum into the stomach. This is produced by spasm of the pylorus and is readily overcome by making steady but gentle traction; or should the resistance prove to be considerable, the clutch is made to release by having the patient drink a little carbonated water. As the bucket is drawn upwards through the cardia, there is felt a noticeable retardation of no importance. At the cricoid region there is usually considerable spasm, but this is instantly relieved by directing the patient to swallow. The act of deglutition relaxes the spasm, whereupon the bucket slips into the pharynx and is withdrawn.

Upon examination of the thread, that portion which has entered the duodenum will be found bile-stained; adjoining this, for a distance of from 2 to 4 cm., the thread shows a characteristic stain where it has been held in the pylorus; above this it may have the natural color. When there is present a peptic ulcer there is a distinct, reddish brown stain corresponding with the diameter of the ulcer and due to the coloring matter of blood. thread lies tense without change in position for several consecutive hours across the open lesion. This produces the stain. In cases subsequently operated upon, the size and location of the ulcer have corresponded with the stain upon the thread. By those who have used the test in a large number of cases it is held that with reasonable uniformity it indicates an open ulcer if present in the duodenum, the pyloric region, along the lesser curvature of the stomach, or at the cardia. It may not give evidence of an ulcer along the greater curvature.

Should the thread be permitted to pass too far into the duodenum, its tension becomes considerable probably from duodenal peristalsis. Should the patient inadvertently swallow the full length of the string, it will be passed with

a stool after a day or two without inconvenience. Even when a bucket is not attached, a string slowly swallowed readily finds its way through the alimentary canal. Taking advantage of this fact, Sippy directs that a string be swallowed, sufficient in length to become firmly anchored in the intestine, where it is held by the convolutions of the intestine and by its peristaltic waves, so that upward traction on the string may render it taut. In this position it serves as a guide for the introduction of a threaded wire or other instrument in case of stenosis in the esophagus, stomach or pylorus.

By utilizing the same method, a narrow rubber tube may be made to pass the pylorus. Through the tube may be withdrawn duodenal contents which may be used for the study of the duodenal secretion and the secretions of the liver and pancreas; or at the end of the tube may be attached a dilatable rubber bag, which upon inflation may serve as an obturator for the duodenum or the pylorus. It may be of service by holding in situ a bismuth mixture when it is desired to study by radiography the caliber and conformation of the part.

Work of this kind has been found practicable by Einhorn and Gregory Cole. The principle involved in the string test has various possible applications, not the least important of which is "duodenal feeding," a process which we owe to Dr. Einhorn and which is hereafter to be described.

## CHAPTER VI

### DUODENAL ALIMENTATION

Through the ingenious contrivance of Dr. Einhorn it is possible to nourish a patient by direct duodenal feeding, thus sparing the stomach and allowing it physiologic rest. The large number of cases in which the method has been successfully employed and the careful clinical study of the subject by Dr. Einhorn, Dr. W. Gerry Morgan of Washington and others both in this country and abroad, warrant the employment of duodenal alimentation in a large group of cases.

The apparatus consists of a rubber tube about the diameter of a chicken quill (4 mm.) and one meter in length. The distal extremity is provided with a conical, perforated bucket about 1 cm. in diameter. The bucket with the tube is swallowed to the distance of about 70 cm. from the incisor teeth, and secured by a loop carried around the ear. Usually within twelve hours the bucket passes through the pylorus. This fact is determined by withdrawing fluid through the tube by means of a suitable glass syringe. Should the bucket be present in the stomach, the syringe readily fills with material characteristic of stomach contents. If the bucket has passed the pylorus, the syringe fills more slowly and the material withdrawn is alkaline in reaction, bile-stained and contains the pancreatic ferments.

When the instrument is known to be in place it is left undisturbed. At intervals of two hours, fluid nourishment at the body temperature is slowly pumped through the tube. About 300 c.c. of a carefully strained solution of egg and lactose in milk is used at a feeding. Following this about 10 c.c. of pure water is passed in for the purpose of clearing the tube, and finally a sufficient amount of air to free the tube from all fluid. The proximal end is then closed.

This practice may be carried out eight or nine times a day for a period of two or three weeks. The digestion is usually sufficient and patients gain in weight.

The limitations of duodenal feeding are not yet known, but the method is especially useful when the pylorus is irritable, as in peptic ulcer. It has succeeded in hastening the cure of many cases of gastric and duodenal ulcer, with and without hemorrhage. Gastrectasis and other conditions attended with ischochymia have made striking improvement under this treatment.

Beck of Baltimore has found the apparatus useful when introducing large doses of ipecac in the treatment of dysentery. Other substances, the presence of which is undesirable in the stomach, may be taken thus directly into the duodenum. The method opens a new field both in alimentation and medication.

My observations lead me to conclude that by its use some cases are curable which otherwise would require surgical relief.

The technique is easily acquired; it needs only conscientious attention to a few details to avoid clogging or over-distending the tube, or chilling or over-heating the duodenum. The duodenum is found to be particularly sensitive to variations in temperature. Also it is necessary to give great attention to the character of the aliment that is introduced by this method. The nature of the food must be varied to suit the individual case. Heedlessness as to details will result in exciting irritation of the du-

odenum, when the tube will be ejected. In case, through mistake in attention to these details, the duodenum becomes intolerant, vomiting will be excited and a return to duodenal feeding must be temporarily postponed. My experience with this method has been successful and I regard it as marking a decided advance in the treatment of a variety of gastric and duodenal affections.

## CHAPTER VII

# RADIOGRAPHY AND FLUOROSCOPY

We are indebted to the admirable work of Cannon, undertaken, he says, at the suggestion of Bowditch, for the adaptation of radiographic shadows to the representation of the position and movement of the alimentary canal.

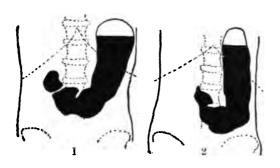
Not long after Cannon's publication numerous foreign investigators applied the method in pathologic study at clinic. We owe much to the contribution of Holzknecht, Groedel, Rieder, Kaestle and Rosenthal.

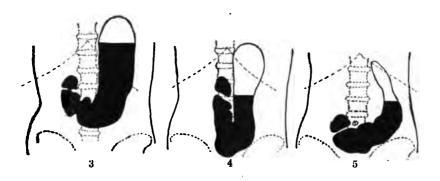
Various expedients have been adopted in producing shadows that are most significant. When unmixed bismuth is swallowed in amount sufficient to represent by shadow the totality of the stomach, so much is required as to become a serious embarrassment if not peril; therefore, the expedient is adopted of giving bismuth with some vehicle, in little masses like pills, or in an emulsion with oatmeal gruel, or in other suitable media like buttermilk. Quite unlike this is a method recommended first, I believe, by Rieder—that of obtaining graphic records without the use of bismuth and a shadow, but by distending the part with gas and obtaining by the Roentgen rays a clear, relatively shadowless field, corresponding with the organ in question. The advantage claimed for the method last described, is that it does not lead to displacement of viscera through weight. In the case of bismuth, when 100 gms. or more are introduced, together with 500 c.e. or more, of water, gravity leads to a considerable weighing down of the stomach, for which allowance must be made in forming a conclusion. (Radiograms I and II.) In studying the colon by means of Roentgen rays, there is ordinarily injected about 120 gms. of bismuth in 1000 c.c. of fluid. When the patient is in the upright position, the weight of this clyster is sufficient to cause a downward displacement of the colon, depending somewhat upon the width and stretching of the mesentery. When the stomach and colon are strongly attached and when there is adequate intra-abdominal pressure, the displacement referred to is of little consequence as to Roentgen-ray results, especially when the patient is in the prone position, yet a certain descent of the organs will be noted when the patient is standing.

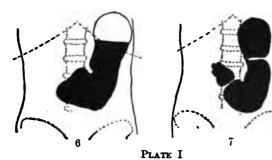
In the case of weakness in the muscle layers of the stomach and colon, or when the viscera too readily undergo displacement downward, these features are at once recognizable in the bismuth shadow. In this there will be found an advantage to offset the objection, for it affords an additional item in the diagnosis.

It has been suggested that the form assumed by the stomach and its rhythmic movements may undergo unusual modifications when submitted to the effect of large quantities of bismuth, besides the effect of the undue weight. There may be ground for the contention, but as yet we are not prepared to estimate such modifications, even though granting their occurrence.

Taken as a whole the radiographic examination is a long step in advance, an almost indispensable measure in the diagnosis of many conditions of the stomach. A feature of X-ray representations that attracted immediate attention was the unexpected range of gastric movements. In the normal stomach under physiologic activity from stage to stage, almost from movement to







TYPES OF STOMACHS

## DRAWINGS REPRODUCING RADIOGRAMS

- Common form of stomach.
   Fish-hook stomach.
   Stocking-leg stomach.
   Water-trap stomach.

- 5. Steer-horn stomach.6. Boot-leg stomach.7. Bilocular stomach.

.  movement of digestive activity, surprising changes were observed in the shape of the organ. Correspondingly remarkable modifications of the shadow, indicative of pathologic conditions, likewise were noticed. Also, the position of the stomach, in relation to other abdominal viscera was found to be less constant than we had believed. At once it was noted that, in presumably normal individuals, certain striking characteristics in form and position persisted, so that it became possible to classify stomachs into types; hence, there are described the horizontal, vertical, 'oblique, "boot-leg," "stocking-foot," "fish-hook," "steer-horn" and "water-trap" types, not to mention other still more fancifully named.

Under the circumstances some misconception was inevitable, and the correct interpretation of radiograms became of fresh interest and added a new importance to the work. Hitherto all effort was expended on perfection in technique; now it was perceived that equal study was needed in interpretation. The modern Roentgenologist must be more than a skillful photographer and technician; he must possess wide knowledge of pathologic conditions and be able to interpret to clinicians the pathologic significance of the radiograms and fluoroscopic findings. In other words, he must be a medical specialist.

Owing to the danger of personal injury in operating the apparatus, the fluoroscopic method came to be superseded by the radiographic. The use of the fluoroscope, however, was never abandoned entirely and, owing to improvements in construction, the instrument is largely employed.

Each method has special advantages and neither should be dispensed with. At length we are procuring in this country satisfactory results from the fluoroscope, and this will lead to the elimination of certain errors in interpretation that, with the radiogram; were almost inevitable. A radiographic picture may be accepted as a correct instantaneous representation of the stomach, yet it may not hint at the shape which the stomach will assume in another moment.

Even with radiograms often repeated in series, useful at times as the method is, it does not equal the fluoroscope in reproducing a succession of events. Recently Kaestle, Rieder and Rosenthal have perfected an apparatus by means of which they are enabled to make cinematographic reproductions of the bismuth shadow. They have succeeded in producing twelve radiograms in twenty-four seconds, thereby greatly increasing the usefulness of radiography by making possible a precise record of moving shadows.

This knowledge of the movement of the stomach is of great value for, when the plates are properly interpreted, it expedites a diagnosis of those numerous and important motor disturbances which appertain alike to functional and structural disease and which comprise the sources of aggravating symptoms.

Another method of studying the movements of the stomach is by utilizing polygrams as suggested by Levy-Dorn. This consists in taking on the same plate, at intervals of 3 to 5 seconds, two or three instantaneous views of the stomach. Those parts of the stomach which have changed in position during the intervals are shown by areas of lighter shadow, which contrast with the deep opacity of the regions which have undergone no displacement. By this plan one is able to obtain a graphic impression of the peristaltic waves and other movements of the stomach in the order in which they occur.

Some interesting results have been obtained by utiliz-

ing the stereoscope. The method consists in taking at brief intervals two successive reproductions of an image. At other times the two impressions are made with the rays falling in slightly different directions. Such plates viewed through the stereoscope give an impression of relief and of depth not seen in other methods of examination.

Transient biloculation from spasm gives a radiogram that reduplicates that produced by organic biloculation from stenosis. They are to be differentiated in recognizing that the shadow representing the contraction in each séance, persists for weeks or months in case of true hour-glass stenosis; whereas, in case of spasm the shadow that it produces is comparatively intermittent; also, in spasm, the contents of the lower pouch can be made, in part, to return to the upper pouch, by causing the patient to lie down, or by manipulating the abdomen over the lower pouch while the patient is in the Trendelenburg position.

It should be remembered that there may be both stenosis and spasm, or long-continued spasm, at first without stenosis, may be excited by a lesion that ultimately ends in stenosis. In such a case the increasing duration of time in which bismuth fails to pass the pylorus is suggestive of the intervention of stenosis.

Double Capsule Means of Recognizing Slow Motion.—Specially prepared capsules insoluble in gastric juice are required. One capsule is completely filled with bismuth and a second capsule is partly filled; then both are swallowed.

The heavier capsule sinks to the bottom of the stomach. That holding only a little bismuth floats, owing to the contained air.

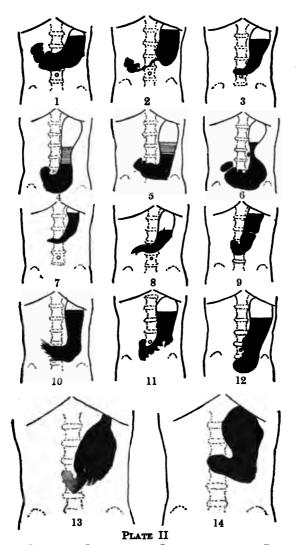
Radiograms then taken show the level of the gastric

contents and also the activity in the evacuation of the stomach.

## THE INTERPRETATION OF RADIOGRAMS

The value of radiograms depends upon their interpretation. At present there is a wide difference between radiographers in the matter of interpretation. On many important points there is a difference of opinion as to the meaning of various shadow representations. knowledge gained from viewing plates needs to be verified by observations made in surgical exploration and by autopsy. Already the subject has gained considerably as a result of these combined methods of study; but much remains to be learned. It is difficult to lay down precise rules for the interpretation of radiograms. They should be considered as yielding important evidence, but a conclusion as to their exact meaning should be formed only after taking into consideration the facts ascertained by other methods of study. The following remarks on the interpretation of radiographic shadows are based upon personal experience and upon the conclusions reached by several eminent American and foreign radiographers.

Normal Stomach.—There are various types of normal stomachs as is clearly shown in X-ray shadows. There is also marked difference in the radiographic appearance of the stomach, during the digestive act and from day to day in the same individual, and this without symptoms. The stomach shadow may occupy an oblique position in relation to the abdomen and whether it takes this, as in the "steer-horn" shape, or one more vertical, as in the "boot-leg" shape, it may be none the less natural and perform its function well. In the study of radiograms that which guides one in deciding that the stomach is normal is largely the manner in which the mo-



SCHEMATIC DRAWINGS ILLUSTRATING INTERPRETATION OF RADIOGRAMS

1. Characteristic duodenal cap.

- 2 Hour-glass or bilocular stomach, non-malignant cancer, showing partial obstruction.
- 3. Hypertonic stomach.
- 4. Dilatation from atony.
- 5. Dilatation from obstruction.
- 6. Diverticulum of stomach from ulcer.
- Small stomach, diffuse cancerous infiltration of walls.
   Small stomach, from excessive motor activity.
- 9. Chronic ulcer, producing deep and prolonged peristaltic contraction.
  10. Adhesions at antrum and pylorus from ulcer or cholecystitis.
- 11. Cancer of stomach, showing ragged edge.
  12. Cancer of stomach.
- 13. Spasm of pylorus and antrum, thirty minutes after bismuth meal.
- 14. Same case, four hours after bismuth meal.



tor function is carried on. During digestion in the healthy stomach, the lower third and sometimes the lower half is seen to contract, assuming a tubular shape and, as it were, grasping its contents. Above this the fundus is seen to have a balloon-like appearance; and its hopperlike action is evident, for a large part of the bismuth meal temporarily rests there until carried down gradually by peristalsis. Above this mass of bismuth at the upper part of the fundus is seen a clear space with a dome-like upper border, corresponding with the form of the diaphragm. This represents the gas which rises above the semi-fluid contents of the fundus. The peristaltic waves in the early part of digestion are seen to begin at the antrum or at a point higher up, and at times the fundus also undergoes band-like constriction as the result of normal peristaltic contraction. The bismuth mixture begins to escape from the stomach soon after its ingestion, entering the first part of the duodenum where it is shown in the characteristic "duodenal cap." This is separated from the stomach, above the end of which it appears to rest, by a sharply defined, clear band which represents the sphincter. As the lower zone of the stomach discharges the bismuth through the pylorus the upper zone, or fundus, gradually empties its contents into the lower zone.

Duodenal Cap.—The importance of peculiarity in the shadow caused by bismuth in the first part of the duodenum is generally recognized. (Plate II, Fig. 1.)

Ordinarily in health there appears a small shadow with a rounded top and a level base, situated above the pyloric end of the stomach and separated from the shadow of this part by a perfectly clear space, which represents the pylorus.

This shadow of the first part of the duodenum looks like a cap sitting above the end of the stomach; from

this appearance it is commonly spoken of as "the duodenal cap." Roentgenologists have found that the shape, position, and density of the shadow of the cap have much significance.

Pyloric Spasm.—The X-ray shadow of the stomach with spasm at the pylorus, depending upon reflex or neuropathic causes, shows no bismuth passing the pylorus. In the antrum pylori, especially toward the pyloric ring, the shadow is thinned out, irregular, furrowed and apparently lined by longitudinal rugae of the mucosa. At times the stomach is in a state of over-tonus, in which case there is little or no evidence of peristaltic waves. (Plate II, Fig. 13.)

Obstruction of the Pylorus.—In obstruction of the pylorus, whether functional or structural, the bismuth appears to be pressed into the antrum pylori, ending abruptly at the sphincter, the bismuth shadow making a sharp edge of demarcation against the clear space above which represents the duodenum. (Plate II, Fig. 14.) There is, of course, absence of the duodenal cap. When obstruction is accompanied by perigastric adhesions or scars that change the contour of the stomach, or when perigastric inflammatory tissue or diffuse infiltration contract it, the outline of the shadow shows corresponding irregularities. (Plate II, Figs. 10–11.)

Adhesions from Pericholecystitis.—When evidence of pyloric obstruction occurs in a patient who has suffered from cholecystitis, there are often deformities of the lower end of the stomach, the result of adhesions which make traction upon the gastric tissues; there may be interruption of normal peristalsis; the antrum pylori may show overfilling, no bismuth escaping, as a result of pyloric spasm. These effects are represented in the X-ray shadow by stopping of the peristaltic wave at the place

of adhesions and by irregularities of outline varied in character, the result of adhesions and fibrous contractions. When there is marked pyloric spasm, the shadow ends at the sphincter. When the closure is incomplete the bismuth may be seen passing in an irregular stream through the pylorus, making an imperfect, distorted and displaced duodenal cap.

Over-Tonic Stomach.—At times from pyloric irritation or other causes, the stomach becomes over-tonic. It is then apparently elongated; and often narrow in its median zone, the two walls of the stomach approaching each other so closely as to suggest biloculation. There is little or no evidence of peristalsis. (Plate II, Fig. 3.)

Irritable Stomach.—When from functional or other causes the stomach is in a state of irritability, the motor functions may be exaggerated, which in the radiogram is shown by the increased number and depth of the peristaltic waves. At times a wave appears almost to bisect the organ by ring-like contraction. Occasionally in the median zone of the stomach these contractions are so marked that the organ is divided into two distinct pouches, giving the picture of hour-glass stomach. These contractions, however, while lasting longer than those of normal peristalsis, are generally less continuous than in case of spasm from ulcer; they must not be mistaken for contraction caused by scar. By a sufficient number of serial radiograms one can occasionally note that the stomach is in comparative relaxation, or that the contractions appear and disappear from point to point, moving onward toward the pylorus. The sphincter is intermittently opened and bismuth escapes into the duodenum where it may be seen in the duodenal cap.

Atonic Stomach.—The radiogram of the atonic stomach shows the lower end of the organ distended. There

is absence of the tube-like lower strait which is present during digestion in the normal stomach. The organ has much the appearance of the stomach seen post mortem. The peristaltic waves are absent, or are represented by scarcely-to-be-perceived notches, representing very feeble waves. The bismuth is not held well in the fundus as is the case in the normal stomach, but sinks by gravity to the most dependent portion. A small stream of bismuth is seen to be escaping through the pylorus scarcely filling the duodenum. There is scanty filling of the duodenal cap. (Plate II, Fig. 4.)

Gastrectasis.—In atonic gastrectasis the radiograms present the appearance of those of a stomach in atony, except that the shadow is larger and extends to the lower part of the abdomen. Very often gastrectasis is associated with ptosis, in which case the shadow is seen for the most part, sometimes altogether, to the left of the median line and its lower border may reach into the pelvis. In some cases of profound atony with dilatation, the shadow is seen in the lower part of the abdomen, while the upper left quadrant is occupied by a large clear space which represents the huge air bubble or collection of gas above the bismuth.

Gastric Ulcer.—In the earlier stages of ulcer, the radiographic showings are only suggestive and must be squared constantly with the facts derived from the history of the case and the course of the disease. In the late stages there often develop conditions that give more precise radiographic findings.

• Spasm of a localized band of muscle fibers, intermittent but persisting longer than the contraction of normal peristalsis, may be observed. This spastic biloculation is formed by a narrow band of contraction that involves the greater curvature and leaves the lesser curve, which is apparently held in place by the gastro-hepatic omentum without even a dent, a characteristic which is likewise true of some cases of organic stricture of the stomach. (Plate II, Fig. 9.) This sign is more diagnostic when it is accompanied by a point of circumscribed tenderness on pressure, and the other clinical evidences of ulcer. It is more significant when the contracture recurs incessantly at the same place, although occasionally disappearing completely; also when, as sometimes happens, it is entirely obliterated during the effect of a hypodermic injection of atropin. Probably adrenalin would produce the same result but so far as I know the trial has not been made.

The occurrence of this spasmodic band is analogous to the rectal spasm which is produced by anal fissure.

However, localized spasm of the stomach, albeit usually less constant in location, may result from neuropathic states when no ulcer is present.

At times there is an exaggerated peristalsis, perhaps with spasm of the pylorus, denoting an irritable state of the stomach. At times there is unusual tonicity with but slight evidence of peristalsis, the stomach being small and giving a shadow that changes but slightly until the tonus subsides.

A spot of shadow remaining after the disappearance of shadow from surrounding parts, was long ago reported by Hemmeter as indicative of ulcer. Although others have noted this, it is generally conceded to be a rare manifestation. Indeed there is sometimes observed to be a "fault" in the shadow corresponding with the site of the ulcer. Neither of these can be regarded as reliable but merely as possible evidences of ulcer.

An interesting peculiarity denoting a strictly localized weakening of the muscular tunics as a result of ulcer has been described by Haudeck and its reality confirmed by the observation of Schmieden, A. Béclère and others. This consists in the appearance of a small, distinct shadow, usually along the lesser curvature, separated from the main shadow of the stomach. It gives the effect of a tiny diverticulum filled with bismuth. (Plate II, Fig. 6.) It may be no larger than a filbert. In order to bring out the separate shadow the stomach needs to be well filled with bismuth mixture. Sometimes the diverticulum is incompletely filled, as is shown by a superimposed clear spot, "air bubble," above the shadow.

The small diverticulum here described may be represented not by a separate little shadow, but, when the bismuth mixture is continuous from stomach to diverticulum, the shadow may take the shape of a little nodule resting on the stomach, or attached thereto by a pedicle. When quite separate from the main shadow it may remain for some time after the rest of the stomach is empty.

The stomach is often constricted by a contraction of the greater curve at a point opposite the diverticulum. Marked tenderness and at times localized resistance may be discovered on palpation over the diverticulum.

**Duodenal Ulcer.**—S. Kreusfuchs <sup>1</sup> states that in duodenal ulcer the stomach at first rapidly empties bismuth contents into the duodenum.

Bergman<sup>2</sup> states that the pylorus is at times insufficient and at other times in a state of spastic closure in duodenal ulcer a view which conforms with my experience. Recently Friedenwald and Baetjer<sup>3</sup> affirm that early pyloric insufficiency is the rule, except when the motor function is still further disturbed because of adhesions or other factors.

<sup>&</sup>lt;sup>1</sup> Deut. Med. Woch., Nov., 1912, No. 46.

<sup>&</sup>lt;sup>2</sup> Münch. Med. Woch., Jan., 1913, No. 4.

<sup>8</sup> Association American Physicians, 1913.

In some cases of duodenal ulcer the bismuth is discharged quickly from the antrum pylori into the intestine, the reverse of what occurs in gastric ulcer. In what proportion of cases this immediate discharge holds true is unknown. Later on, the discharge ceases for a time when the X-ray shadow is indistinguishable from that produced by gastric ulcer. In order that this peculiarity in the motor function should be most readily observable, not only should the radiogram be taken immediately after the bismuth meal but the bismuth mixture must be given when the stomach is known to be empty. The presence of even a small amount of aliment is sufficient to prevent the movement here described, the antrum then retaining the bismuth.

Friedenwald and Baetjer have noted that after the bismuth has entered the duodenum there is found a small, clear spot at the site of the ulcer, showing no shadow or an imperfect one. Duodenal ulcer in a fair number of cases gives rise to an inflammatory thickening of the pyloric end of the stomach, to stenosis and spasm of the pylorus, conditions which retard the escape of the bismuth mixture and which give a shadow not to be distinguished from that of pyloric ulcer. This was true of case XVI, page 300, as is shown in the description of the radiogram.

When there is constriction of the duodenum, the part above is dilated, sometimes extremely so; it has even been reported that antistalsis is at times discernible.

Cole says that when the duodenal cap is in position but is contracted and has a worm-eaten appearance and when the duodenal surface of the sphincter of the pylorus is irregular, duodenal ulcer should be considered.

The radiographic evidence of duodenal ulcer has not yet reached sufficient perfection to warrant making a

diagnosis on it alone. It contributes information that is at times most valuable, but results are rendered uncertain because of the effect upon the shadow of adhesions, outside pressure, coincident gastric ulcer or other stomach disease and because of accidents of a technical character.

Cancer of the Stomach.—When cancer invades the walls of the stomach the radiograms differ widely in different cases, depending partly upon the location of the growth, partly upon the degree of encroachment upon the gastric cavity and partly upon irregular thickening of the walls due to infiltration. The shadow, therefore, is at times irregular, undulated, and sharply defined. Certain regions of the stomach may appear to be cut out; at other times the border of the shadow is thin, having a ragged and torn edge, and sometimes an appearance well characterized by the term "worm-eaten." (Plate II, Fig. 11.) As a result of involvement of the serous coat of the stomach with resulting adhesions, the position of the stomach becomes unnatural and this is shown in the radiogram. The peristaltic waves halt at the beginning of the tumor.

The Roentgen rays should be engaged in the routine study of gastro-intestinal diseases; indeed, a diagnosis of any but the simple cases is incomplete without the evidence thus obtained.

## CHAPTER VIII

#### DYSPEPSIA

In adopting the term which heads this article. I am not unmindful of its frequent misuse nor of the questionable pathology which, from time to time, parades under the name. The term was formerly used in the description of a large group of cases in which gastric symptoms constituted the chief complaint but in which no physical basis of disease was discovered. There came a time when great importance was ascribed to the digestive ferments, and their supposed deficiency was believed to cause the various forms of gastric distress; and so it came to pass that the old name dyspepsia was replaced by the term indigestion, and for years the treatment of the condition was limited mostly to the administration of pepsin and hydrochloric acid. With a wider knowledge of the chemistry of the stomach in health and disease, which followed the use of the stomach tube and the minute examination of gastric contents, it was discovered that in some instances patients were suffering, not from indigestion, as the term had been understood, but rather from over-digestion, hyperpepsia and oversecretion. At length the term indigestion fell largely into disuse and, with the advent of those who with the utmost detail studied the variations of digestion clinically as well as experimentally in animals, there grew up the conception of many special derangements of the stomach which were referred to peculiarities in functional activity. Hence arose the classification depending upon

disorders of secretion, motion and sensation. When, from any cause one of these was deranged, such derangement was regarded as a more or less distinct affection. The terms hyperchlorhydria, hypochlorhydria, intermittent secretion, gastric unrest or spasm, motor insufficiency, atony, hyperesthesia and many others were employed to describe these conditions. It cannot be denied that this view of the pathology of the stomach has proved illuminating and that it has led to a great advance in the understanding and management of the affections in question. It still remains, and probably will remain, necessary to give attention to individual abnormalities in the large group of manifestations related to disturbed gastric digestion. For that reason I have devoted some space to the special consideration of these conditions under the heading. The Functional Neuroses. However, it must be admitted that most of these disorders do not represent clinical entities. Sometimes they are dominant features in a case and recovery is delayed until they are corrected: that is, until there is controlled a hyperchlorhydria, a motor excitability, or some unusual sensory condition. But although this is true, we recognize that most patients cannot be relieved merely by the correction of a single manifestation of gastric disturbance. Such patients suffer from difficult or painful digestion; they speak of themselves as dyspeptics, and it is proper to describe the condition as dyspepsia or difficult digestion.

The explanation of dyspepsia is not the easy task which we have sometimes deluded ourselves into believing. The problem varies with the temperament of patients, with their habits, environmental peculiarities and with the differences in exciting cause. A convenient explanation of the symptoms has been to regard them as the expression of gastritis, a pathological notion that had

sway for generations. Following this came the doctrine that the symptoms resulted, not from any structural change in the stomach, but as the manifestation of reflexes from nerve irritation. Original investigations have been made, brilliant writing has been done and there has been an immense amount of case report\... The partisans of each of these theories have found in these facts explanation for about all that is important in the nature of dyspepsia. The source of the reflex nervous irritation by the gynecologist, was discovered in the female pelvic organs; by the genito-urinary surgeon, in deep urethral stricture; by the neurologist, in spinal irritation and psychopathy: and by the oculist, in eye-strain, resulting from errors in refraction or from muscle unbalance or both. satisfied that no unprejudiced clinician who has openmindedly studied the question is able to deny that a large number of dyspeptics are relieved by the removal of irritation, acting reflexly through nerve paths. Nervous disharmony is undoubtedly at times responsible for dyspeptic symptoms, yet all cases of dyspepsia are not cured by the amelioration of a focus of irritation. No attempt is here made to catalog the sources which may produce the various forms of gastric distress through the intervention of nerve irritation. Some are more important than others, for instance, the gall-bladder, the appendix and the eves, and each of these are worthy of special consideration. It is proper to add that in the case of the gall-bladder, the appendix, the colon, the urinary bladder, etc., the explanation is not limited to reflex nervous causes but includes also the direct and indirect results of infection, including the element of intoxication.

Before taking up the consideration of toxic and other causes that may be complicated with nervous disturbance

in producing dyspepsia, attention is directed to the influence of special divisions of nerves.

### VAGOTONIC AND SYMPATHETICOTONIC STATES

Much interest has been awakened in the nature of functional disturbances of digestion by the recent contributions to the physiology of the nervous system. It has been pointed out that a physiologic antagonism exists between two distinct divisions of ganglionic nerves. One of these divisions, called by Langley the autonomic system of nerves, is opposed in its action to that of what is now regarded as being truly of the sympathetic system. Those nerves affecting the abdominal viscera and belonging to the autonomic system, are represented by branches of the vagus.

It is held, particularly by Eppinger and Hess, of Vienna, that in certain individuals especially, and in all individuals under special stimulation, the vagus branches are dominant in action, thus disturbing the balance. An organ affected by this disturbance of balance is said to be "vagotonic," and an organ affected by a disturbance of balance caused by dominant action of the sympathetic, is spoken of as "sympatheticotonic."

The stomach in a vagotonic state responds to slight stimulation by increased muscular tonus, at times by unusually strong peristalsis, also by strong or even spastic closure of the pylorus. The gastric secretion becomes unusually active and the stomach contents may have a high acidity. The increased tension of and the increased secretion by the stomach are often attended by sensations of discomfort or distress.

It will be noted that these several conditions are those that formerly were described as manifestation of an *irritative neurosis*, while symptoms of an opposite character were known as the expression of a depressive neurosis. Illustrations of the latter appear in instances of atony, hyposecretion and anorexia. The contributions of von Noorden, Eppinger and Hess and others throw light upon the mechanism of the gastric neuroses but they do not materially increase our practical knowledge of the subject. It is true that the nomenclature is undergoing modification and certain conditions, as pyloric spasm, etc., are coming to be spoken of as vagotonic states, while functional atony, etc., are mentioned as sympatheticotonic states. However, clinically we recognized these states long ago but gave to them other names.

Doubtless a real advance in our conception of the gastric neuroses has been made by the pharmacologic studies which show that certain drugs have a distinct vagotonic or vagus stimulant action while others have a paralyzing effect upon the vagus branches; certain other drugs have a sympatheticotonic stimulant action and probably there are those that have a depressing effect especially on the sympathetics.

The selective actions of each of several drugs upon the autonomic and upon the sympathetic system of nerves respectively, have been studied and tabulated, notably by von Noorden, Jr., by Eppinger and Hess and in this country by Barker and others.

It is manifest that the drugs so far used, although their action lends support to the doctrine of an antagonism between the autonomic and the sympathetic nerves, are too complex in action for satisfactory demonstration of specific effects. Some drugs, as atropin, act upon one portion of the autonomic system and upon the remainder but slightly or not at all. Other drugs, like digitalis, produce results that are too complex to permit of classifi-

cation. So far as relates to practical work on the stomach, it is shown that atropin, by depressing the vagus fibers decreases the motor and secretory functions; and that adrenalin, by stimulating the sympathetics, produces much the same effect.

Although physostigmin and some other drugs definitely stimulate the autonomic system, we do not yet possess an agent that can be relied upon to energize that part of the vagus which especially arouses motion and secretion in the stomach.

Certain individuals, according to Eppinger and Hess, are naturally "vagotonic"; others, "sympatheticotonic." In these the selective action of drugs is exaggerated; that is, some are particularly susceptible to the effect of atropin, some to pilocarpin and so on.

There is much yet to learn of the fascinating subject of visceral innervation before laws can be specifically applied in therapeutics; evidently the knowledge so far available is confirmatory of the pathology of gastric neuroses that already had been accepted by clinicians.

For a long time there was carried on in France and later in other countries an instructive discussion on the origin of dyspeptic symptoms. One group of physicians, among whom Germain Sée was conspicuous, held that the nervous system was responsible; another group, represented by Prof. Hayem and his followers, saw in these symptoms little save the expression of gastritis varying in kind and intensity but almost omnipresent, sometimes even without inducing symptoms. The first group maintained that dyspeptic patients generally presented no evidence of any structural lesions whatever. The other group insisted that indubitable signs of gastritis were nearly always present and that the fault lay in the in-

ability to recognize them. By them was made a refinement of differentiation between parenchymatous and interstitial gastritis, between acid gastritis and gastritis with suppression of secretion that was complex but not altogether satisfying. The contention between the advocates of these two widely different theories of dyspepsia proved advantageous in that it led many clinicians to make independent investigations. Yet to-day there is not perfect agreement in the profession on this vexed question. I have long been convinced that in many dyspeptics there exists no ascertainable structural change in the stomach, for very often it is possible to discover at some remote point a local irritation the removal of which is followed by relief of stomach symptoms. Therefore, I am unhesitatingly committed to the doctrine that dyspeptic symptoms are often the result merely of nervous disturbance which in some way brings about gastric distress. On the other hand it must be admitted that many cases supposedly neuropathic or excited perhaps by disease of some part not directly related to the stomach, are suffering from irritation of the gastric mucosa and that, in some instances, actual gastritis is present. The only conclusion is that symptoms almost identical, may be induced by disturbance of the nervous system, by auto-intoxication, by metabolic failure or by gastritis. Not infrequently several influences act simultaneously to produce the symptoms complex. In the question of dyspeptic symptoms secondary to toxic causes, we enter a field of many possibilities, some of them as yet rather vague. We have to consider the possible results of the offensive or toxic by-products of fermentation, but the question of alimentary intoxication extends beyond this. It may be well to approach it from the standpoint of the so-called idiosyncrasies.

## FOOD POISONING, AUTO-INTOXICATION, ANAPHY-LAXIS

The real nature of idiosyncrasy is not clear. It was long attributed to metabolic peculiarities and, somewhat later, gastro-intestinal auto-intoxication was brought forward as the special baneful factor. It was argued, not without some laboratory proof, that the evil effects following the ingestion of certain articles of food owe their origin to the development of very toxic substances in the digestive tract. Most clinicians have felt that serious intoxications from the intestinal tract arise from the action of intestinal bacteria. Indeed, it is a well-established fact that fermentation changes do give rise to the formation of toxic substances that induce symptoms of more or less gravity. As yet it is not possible to estimate the precise rôle of bacteria in exciting auto-intoxication, toxemias and their train of digestive and constitutional symptoms. Certain foods have acquired an evil reputation for inducing attacks of so-called auto-intoxication.

The subject of food poisoning has grown in importance of late years and upon it considerable light has been shed through the contribution of Finkelstein, brought to notice through the work of Irving M. Snow. Finkelstein, having studied the subject especially in children, divides the question of alimentary intoxication into four stages. First, a disturbance of balance in which there is lessened tolerance with a limited diet in a normal child, and in which an increase or variation of food will lead to digestive disturbance and loss of weight. A second stage, in which there arises dyspepsia with greatly diminished toleration of food and in which food must be withdrawn. A third stage, in which there is wasting with marked intolerance, especially of fats and sugars. If under these cir-

cumstances the food is increased, there is a marked reaction with disturbance in temperature, respiration and heart action and with great depression. In the fourth stage the more serious effects are manifested.

The whole subject of metabolism is as vet too deep for our sounding. We must admit the toxic effect of bacterial products. We must recognize food intoxication resulting simply from lack of digestive and assimilative power and, finally, we have to consider the possible occurrence of anaphylaxis which would explain the strange intolerance shown by some persons for certain articles of food. A child perfectly healthy when nursed by one woman may become acutely and sometimes alarmingly ill when changed to another nurse. Ingestion of cow's milk may cause similar results. This may happen even with pasteurized milk or with milk of low bacterial count in which any unusual bacterial activity seems improbable. In adults, somewhat similar disturbances may follow the ingestion of cheese, mussels and other shell fish, lobsters, eggs, butter and other articles of food though these are wholesome in the average individual. Formerly these ill effects were attributed to a vague influence termed "idiosyncrasy." Recently the suggestion has been made that these very distressing and sometimes dangerous results from the ingestion of foods that ordinarily are easily digested and wholesome may be dependent upon anaphylaxis. Von Pirquet, Rosenau and others have demonstrated the importance of this principle, which is illustrated especially in the baneful effects of the repetition of subcutaneous or intravenous introduction of certain sera. In these cases the first introduction causes no trouble but a later injection produces marked symp-This has awakened us to the dangers of anaphytoms. This matter is fairly well understood as regards laxis.

the repeated use of sera. The question is, may not somewhat similar reactions arise from taking some toxalbumin which, after absorption, may induce an anaphylaxis corresponding to that produced by sera? Recent experimental work lends support to this doctrine of alimentary anaphylaxis, an hypothesis which clinical observation has made seem more and more probable. The symptoms which develop in patients who are victims of "idiosyncrasy" to certain foods, are strikingly similar to those of the so-called "serum disease."

The clinical manifestations of true anaphylaxis are characteristic and significant. The symptoms are excited by a very small quantity of albuminoid substance. This sudden, almost explosive appearance with a decrease in blood pressure and in body temperature is very characteristic. In both "serum disease" and food "idiosyncrasy" occur the systemic shock, the marked cardiac depression interfering with the movement of the blood, the grave internal congestion, together with dyspnea and asthma, and intense urticaria. Ultimately there may be found some other explanation for idiosyncrasy to food than the principle of anaphylaxis, but there certainly exists a very suggestive similarity between its manifestations and those of serum disease. An identity of cause would explain the food poisoning described by Finkelstein.

What relationship may exist between alimentary intoxication with metabolic disturbances and gastritis? Probably we have in this, aside from infection, a direct cause of local reaction in the gastric mucosa. Edema and swelling of the mucosa as a result of the ingestion of certain foods is undoubted and it is inconceivable that a local reaction producing hyperemia and edema could exist in the stomach, save temporarily, without having infec-

tion follow in its train. Although these results occur as a consequence of unfortunate accidents in eating, it leads one to believe that the less striking but more prominent manifestations of gastric irritation may be induced by the taking of food that is only moderately unsuited to the patient and yet which proves directly and indirectly sufficiently noxious to give rise to local symptoms. not always possible to determine which particular article in the dietary is causing the trouble, but a search for it is worth while. Many times we find that a man loses his dyspepsia when he renounces smoking, or he may be able to smoke one kind of tobacco and not another unless he would suffer the return of his symptoms. A similar story may be told regarding the effect of coffee, tea and such familiar articles of diet as eggs, milk, certain fruits and vegetables that are ordinarily innocuous in their action.

Metabolism and the Liver.—There is a popular belief that some of these disturbances of the stomach are but the secondary results of a derangement of the liver. When we consider the important rôle played by the liver in the processes of digestion and assimilation, it may reasonably be inferred that this organ is responsible for stomach symptoms even when the stomach itself is without sign of disease. Both active and passive congestion of the liver cause gastric hyperemia. Many of the so-called bilious attacks which are preceded and accompanied by dyspeptic symptoms are, we have reason to believe, the result of derangement of the liver. An old acquaintance, yet one which has recently reawakened our interest, is what the French call "la foie torpide" or the lazy liver.

TORPID LIVER.—Torpid liver seems sometimes to depend upon disturbances of innervation and occurs after mental

shock, insomnia or prolonged intense mental activity. At other times it develops after unusual physical exercise, or may occur without assignable reason, somewhat periodically. It is closely related to functional derangement of the intestines, and it rarely appears without its gastric accompaniment. The insufficiency of hepatic secretion may be shown by study of stools and urine. frequently occurs in women at middle life, sometimes in patients having migraine, or eczema and especially it follows general depression. In some cases it seems to be related to lithemia and to that vague state supposed to be dependent upon excess of purin bodies. Sufferers from torpid liver are usually dyspeptics and have nervous symptoms. There is a sensation of fullness about the liver. There may be enterocolitis, often with constipation and frequently with atony of the gastro-intestinal tract and ptosis of the abdominal organs. Such patients are particularly susceptible to disturbance from improper articles of diet and are upset by hearty meals or irritating foods. At the time of these attacks the tongue is heavily coated and the breath has a disagreeable odor, sometimes ammoniacal or sulphurous. The abdomen becomes distended and the patient suffers disagreeable abdominal sensations, sometimes accompanied by nausea and even vomiting. The attacks are often preceded by unusual appetite which is followed by a period of anorexia. Occasionally the abdominal attacks are accompanied by diarrhea alternating with constipation. frequently the constipation persists and is very obstinate, in which event there is apparently an over-tonic state of the intestine. The size of the liver changes abnormally from time to time. That the liver is at one time overactive and at another inactive is shown by the appearance of the stools, which are one day deeply colored and perhaps the next day pale or clay-colored. The stools often contain an excess of mucus and occasionally are hard, twisted and sometimes granular and "sandy."

Hyperemia of the Liver.—There is a form of dyspepsia accompanied by gastric over-acidity which appears to be secondary to hepatic irritation and congestion. It usually follows a period of over-exercise or mental or nervous strain, associated with over-eating and, perhaps, the unaccustomed although moderate use of alcohol. Occasionally the same manifestations occur without any irregularities in life, but preceded by marked increase in appetite for two or three days. There is a feeling of dullness. The bowels are inactive. There is fullness of the hemorrhoidal veins, often a feeling of weight in the right hypochondrium accompanied by acid regurgitations and a burning pain, sometimes severe, in the epigastric region. This is temporarily relieved by large doses of antacids, sodium bicarbonate and bismuth. Often the medication must be repeatedly given before the pain is assuaged. In these cases there is usually no nausea and the most striking symptoms are referable to the gastric over-acidity.

These attacks of gastric distress recur, notwithstanding temporary relief, until the hepatic congestion passes away. Gray powder or calomel in combination with rhubarb and soda should be given early and this should be followed by a full dose of sodium sulphate. Under this treatment the attack may be aborted or quickly cured.

## CHOLECYSTITIS AND DYSPEPSIA

The symptoms of cholecystitis are at times almost entirely referred to the stomach. This is true in mild cases as well as in more serious gall-bladder diseases in which, perhaps, gall-stones are present to increase the irrita-

tion. Even in those very mild cases which on operation show no signs of pericholecystitis, no thickening of the walls of the gall-bladder, no gall-stones and but little that is apparently pathologic, merely an increased viscidity or unnatural color of the contained bile, the stomach symptoms are the preëminent source of complaint. The symptoms are not permanently relieved until the gall-bladder condition is relieved either by a proper course of medical treatment or by operation.

In acute cholecystitis with enlargement of the overlying portion of the right lobe of the liver the gastric symptoms may predominate.

Definite attacks of acute gall-bladder disturbance are followed by symptoms referred particularly to the stomach.

Chronic cholecystitis, without acute exacerbations, may also give rise to gastric symptoms causing the true source of the difficulty to be overlooked. This mistake is easily made on account of the intensity of the symptoms complained of and their location. There is often gastric unrest occasioned by an excited and disturbed motor function of the stomach, a feeling of tension and contraction, sometimes accompanied by a sense of suffocation, which is relieved by the eructation of gas. There may be regurgitation of gastric juice which, because of its bitterness and acidity, is believed by the patient to be an evidence of stomach disease. The appetite is usually irregular, often there is anorexia, at other times a craving for food with distress after eating.

The examination of the stomach contents at such times usually shows an over-secretion of gastric juice with hyperacidity. This is in part owing to a sympathetic irritation of the stomach without spasm of the pylorus, but in a proportion of cases pyloric spasm is an important

manifestation and, when present, contributes to the hyperacidity. With this condition besides gastric distress there is sometimes intense gastralgia. Occasionally no other symptoms are encountered except paroxysms of intense pain located just below the ensiform process. These recurrent attacks of gastralgia with normal stomach contents may find explanation by examination of the hepatic region and may not be relieved until the gallbladder is drained. Occasionally the pyloric spasm is so intense that food stagnation occurs. In such instances it is not easy to differentiate between cholecystitis and ulcer at the pylorus. The question is made more difficult because the intense hyperemia which sometimes accompanies the condition may lead to slight hemorrhage into the stomach, and therefore we find in the contents, red blood cells or occult blood, and the latter may be found in the A differential diagnosis may require much study stools. of the case.

Operation is inadvisable in such cases until they have received painstaking and expert medical treatment; for although eventually complete relief may not be obtained without operation, this is not always so and, at least, a reasonable time taken for the study of the case will not jeopardize the interest of the patient, and may avert operation. Undoubtedly cases supposed to depend upon obstruction at the pylorus are subjected to gastro-enterostomy without great relief, the reason being that the gall-bladder continues to excite the stomach; obviously, however, a skillful surgeon's observation of the biliary structure should prevent a useless anastomosis.

Treatment.—Patients suffering from cholecystitis are often greatly relieved of their dyspepsia and indeed of all the other symptoms of cholecystitis by the skillful practice of lavage with hot normal salt solution, the use

of alkaline and sedative mixtures, a plain diet and the drinking of an alkaline-saline water, an hour or more before breakfast each morning.

Magnesium salicylate 0.60 gm. (gr. x) or aspirin 0.15 gm. (gr. ii ss) repeated three times daily, either alone or associated with eunatrol 0.15 gm. (gr. ii ss) and phenolphthalein, 0.15 gm. (gr. ii ss) frequently promote the patient's comfort and facilitate the cure.

## CECAL STASIS, "CECUM MOBILE" AND DYSPEPSIA

The alimentary canal possesses two pouches or reservoirs: the stomach, which receives and for a while retains the food in the beginning of digestion; the cecum, which receives and for a while retains the remnants of digestion when, for the most part, digestion is completed. In either of these there may occur motor insufficiency, either from atony or from over-tonicity. The cecum normally retains the contents in order that the imperfectly digested particles may undergo further separation, hydration and absorption. The intestinal content gains in consistency, thus offering mechanical stimulation to the cecum, and expulsive peristaltic waves move the mass upward into the ascending colon.

The intestinal bacteria are very abundant and active in the alkaline medium of the cecum and, as a result, putrefactive changes in its contents may proceed rapidly when there is an unnatural delay.

Motor insufficiency of the cecum leading to stagnation of its contents may result from local irritability causing spasm of the outlet of the pouch from disturbed innervation of the part, from narrowing of the ascending colon, from acute bending of its hepatic flexure, or from displacement of the cecum, conditions which have their analog in the causes of gastric motor insufficiency. Over-

motility and displacement of the cecum, to which Haussmann, in 1904, gave the name "cecum mobile," leads to definite clinical manifestations to which attention was first directed by Curschmann. (Radiogram III.) From any of the foregoing conditions sufficient irritation of the part may be induced to exaggerate the expulsive function of the colon and thus produce a transient diarrhea. other times an over-spasticity of the bowels develops, retarding the onward movement of the contents and causing constipation. This stasis may be accompanied by an irritability of the lower colon with frequent small dejecta, perhaps mucoid in nature, or by retarding spasticity; there may be constipation with a sense of heat and discomfort in the rectum and distension of the hemorrhoidal veins. There arises at the same time a fullness of the portal vessels, with increase in the bulk of the liver with attending hyperemia and slight tenderness. The effect upon the stomach, the primary digestion as a whole and the general economy is marked and inevitable.

New seizures are favored by a state of chronic irritability of the colon and cecum due to the preceding attacks and the parts never quite recover from the effect of one attack of stasis before another develops. Enteroptosis with a general dragging of the abdominal organs upon their mesenteric attachments, lowered intra-abdominal pressure and lax abdominal wall, predisposes to cecal stasis. This is particularly true when the cecum is displaced, that is, in cases of "cecum mobile." (See Radiograms III and IV.) This condition, first described by Wilrus, is only distinguished from cecal stasis otherwise occasioned by careful physical examination, including radiography. The derangements of primary digestion induced by cecal stasis are frequently and sometimes justly attributed to chronic appendicitis. In this connection it is well to state that

the irritability of the appendix is as often the result of the state of the cecum as the cause of it. Many patients who have undergone appendectomy, and who thereby have been somewhat relieved, nevertheless continue to suffer the effects of coprastasis. On the other hand chronic appendicitis is at times the primary cause, and after appendectomy the tendency to stagnation disappears.

In other cases relief is not obtained without removal by surgery of the so-called veil of Jackson. In a few cases of cecal stasis benefit is reported to have followed the anchoring of the cecum.

Diagnosis of Cecal Stasis.—With the acute attacks there is abdominal pain or cramp, at times fever, a noticeable fullness in the cecal region, tenderness over the abdominal sympathetic ganglia, loss of appetite, gastric distress and motor insufficiency, a heavily coated tongue, foul breath and the general symptoms of auto-intoxication.

CHRONIC CECAL STASIS.—The symptoms thus far described have been those sufficiently local in character to direct attention unmistakably to the intestinal tract.

In the majority of cases the attacks are less definite and the patients complain of the stomach.

The physician is not usually so far misled as to locate the trouble in the stomach, yet this occurs in some cases because of the habitual complaint of dyspepsia and because during the exacerbation of the cecal trouble there is increased gastric derangement. Under these circumstances the physician may reason that there is a descending infection, that the stomach is primarily at fault and the intestine and liver are suffering as a result.

The Course of a Mild Attack of Cecal Stagnation.—
There usually occurs for a day or more a decline in the



Radiogram No. I.—DISPLACEMENT OF STOMACH OCCASIONED BY POSTURE, AND IN PART OWING TO BISMUTH. Prone position.



Radiogram No. II.—DISPLACEMENT OF STOMACH OCCASIONED BY POSTURE, AND IN PART OWING TO BISMUTH. Standing position, same patient, taken five minutes later.



Radiogram No. III.—CECUM MOBILE. GASTROPTOSIS.



Radiogram No. IV.—FROM A CASE OF CECAL STASIS.



amount of the daily alvine evacuation. With this there is a loss of appetite, an increased coating of the tongue and often an unnatural odor to the breath.

In individuals of strongly fixed habits of eating, the appetite continues, but soon eating occasions discomfort and later distress. There is a feeling of abdominal fullness, sometimes a sense of tension or moderate pain over the region of the cecum and the border of the liver. Upon palpation, unusual tension of the abdomen is found, more marked over the cecum and other parts of the colon. The stomach symptoms are anorexia, gastric flatulence, eructations, sour stomach and later nausea and perhaps vomiting; in other words, there is disturbance of the motor function. Meantime, well-known general symptoms appear: lassitude, irritability, headache, mental confusion and mild vertigo. The skin loses its freshness, and the urine often shows an increase of pigment, total acidity and urates.

A systemic reaction with a sick headache, vomiting and, occasionally diarrhea, may terminate the attack.

There is instinctive desire to empty the prima viae and the patient ultimately finds some purgative that suits him and to which he resorts. As before stated, there may be a deceptive diarrhea from the lower part of the colon, but this fails to afford relief. The experience may be repeated so often that the patient becomes familiar with the symptoms and describes them as "stomach attacks," "bilious attacks," or "sick headache." It is possible to mistake the condition for migraine.

Treatment.—When the cause consists of an anatomical defect it is not possible to avoid the attacks unless that defect is removed. When dependent upon disturbed innervation this may be corrected through improvement in general health,—more physical exercise and greater reg-

ularity in living. On the approach of an attack, fasting should be advised, calomel and jalap given in full doses, followed by sodium sulphate or castor oil, 30 c.c. (3i). With some a less active medication will suffice. Physostigma extract, .01 gm. (gr. 1/6), hourly for three doses followed by a Seidlitz powder may be all that is required. In any event an irrigation of the colon with a solution of boric acid or inspissated ox-gall, 8 gm. (3ii) water 2000 c.c. (Oiv), is a timely measure.

In a proportion of cases medical treatment is so unsatisfactory that surgical relief is advisable. The attempt should be made to procure thorough emptying and drainage of the cecum by a permanent opening either by means of appendicostomy or by cecostomy as recommended by Reed. Following this operation I have seen complete relief in some desperate cases.

In certain cases, owing to deformity or faulty position of the cecum and other parts of the colon, the symptoms may recur even after having been relieved by the medical treatment described. Modern surgery seeks to remove this embarrassment permanently by resection of the cecum, or other portions of the colon, so as to remove obstructing angulations and secure better drainage. These somewhat formidable operations are warranted if we may judge from the numerous cases reported, especially by Sir Arbuthnot Lane.

Colon Bacillus Infection.—I have convinced myself that an important factor in the development of stomach symptoms in cecal stasis is the loss of immunity to the colon bacillus which grows so luxuriantly in this condition. In any interruption of the intestinal stream, no matter what its cause, there is resulting intoxication and infection which varies widely in different cases. It appears to me that this variability is merely a question of degree

of immunization, as may be shown by obtaining the op-The bad effects of the colon bacillus in sonic index. patients with lowered immunity is a matter of great importance, and in certain cases this may be demonstrated satisfactorily. Patients with colon bacilluria often suffer from heavily coated tongue, anorexia, gastritis, nausea and vomiting. The temperature becomes subnormal with an occasional brief elevation; there is a loss of clearness and glossiness of the skin, loss in weight, lassitude, mental depression and other nervous symptoms. tory depression and renal inadequacy develop. condition so often referable to infection of the urinary tract occurs also in many cases of coprostasis. striking improvement which follows the thorough cleansing of the alimentary canal depends partly upon relief from the colon infection and partly from the removal of other protein poisons, the action of which has been so well emphasized by Vaughan. (Journ. Am. Med. Assn. Nov. 15, 1913.) This matter is overlooked because the intestine, the natural habitat of the colon bacillus, is involved and it is forgotten that the absence of noxious effects of these bacilli is dependent upon a proper immunity. The point to be urged is that this immunity may be defective, perhaps temporarily, perhaps for a long period of time, during which the patient experiences among other things biliousness, indigestion and the other symptoms heretofore described. Very often the liver or the stomach is accused. The mistake is the more readily made for the reason that the functional power of the stomach is usually compromised, often to a marked degree; but this is an effect and not a cause, and in order to relieve the patient some method of treatment must be invoked aside from that of diet and local treatment of the stomach.

# GASTRIC DISTURBANCES SECONDARY TO CHRONIC APPENDICITIS

Everyone recognizes that anorexia, nausea, vomiting, gastric distress and pain are among the very common symptoms of acute appendicitis. These symptoms in a more persistent form are found in recurring and chronic It is not so well recognized that protracted irritative stomach conditions may depend upon appendicitis not otherwise evidenced. It is true that surgeons have insisted upon this fact and some advise appendectomy for the relief of dyspepsia, almost to the exclusion of medical treatment. It is important that greater clearness of vision and finer discrimination should be practiced in dealing with this subject. It is fallacious to take the ground that appendicitis is so frequently the cause of chronic dyspepsia as is proclaimed by certain oversanguine men. However, there is some truth in the claim. Chronic appendicitis is conspicuous among the abdominal irritations which reflexly or otherwise give rise to the gastric symptoms that are described as chronic dyspepsia. The painstaking student of gastric diseases recognizes that more than 50 per cent of the patients who complain of stomach symptoms and of little else, are found upon examination of the stomach to have no discoverable lesion of that organ. Still further it may often be shown that the physiology of the stomach is very little deranged. In other cases there may be disturbances of motion, secretion or sensation yet no structural change. With such negative findings the old query is made: Where is the disease? And the answer is: In some cases it is in the sympathetic nerves, or in brain irritation and systemic disturbances resulting from overwork, anxiety, insomnia, etc.; or it results from renal insufficiency and intoxications from various sources; and finally, that it results from trouble in organs bearing an important relation to the stomach, and although outside that organ it is, nevertheless, often in the abdominal cavity or pelvis, most often in the gall-bladder, the cecum, or the appendix and in the condition of habitus enteropticus.

In looking over the array of causes of the so-called functional disorders of gastric digestion, it will be seen that it is possible for one, because of peculiar personal experience, a certain mental complexion, or because of exaggerated enthusiasm begotten of experience in a single case, to form a lop-sided opinion concerning the cause of functional gastric disturbance. One etiologic factor is made to fill up the foreground to the exclusion of another equally important but more obscure. For instance, eye-strain is a really important cause in the production of functional gastric disturbances. It most certainly would be fallacious to exclude eve-strain from consideration vet it is equally fallacious to bear eve-strain in mind to the exclusion of consideration of an obscure chronic appendicitis. At the same time, though obscure chronic appendicitis is sometimes the source of dyspepsia, this should not lead us to sanction the rather indiscriminate abdominal sections that are made in the hope of relieving gastric symptoms. I have so often seen stomach trouble continue after removal of an appendix imagined to be at fault that I feel strongly against "jumping at conclusions" in this matter. It has come to be a practice to explore the abdomen for the purpose of diagnosis, when, if nothing wrong is found, the appendix is removed as a routine. If this fails to produce a good result, the gall-bladder is drained. The patient sometimes has temporary relief, probably as the result of

suggestion but later he returns to the general practitioner for treatment of the old symptoms.

The following case is an aggravated instance: was referred to me a middle-aged man who complained of agonizing pain in the epigastrium with gastric distension. This suffering had been so acute that, having failed to find relief by ordinary measures, he had gone to a surgeon who drained the gall-bladder although the organ appeared to be healthy. There was no improvement. Within a year another surgeon opened the abdomen and broke up adhesions. As the suffering continued, the advice of some of the best diagnosticians in Europe was sought; then the patient returned to this country where at a celebrated clinic the appendix and the gallbladder were both removed. The patient was now relieved of his symptoms for a period of six months whereupon they recurred with their former intensity. He still complained and operation was done again, this time to break up all discoverable adhesions and to sew up a hernia at the site of one of the scars. No relief followed this procedure. After long suffering the patient again visited Europe where in Vienna after several consultations a gastrostomy was performed and a rubber tube introduced which the patient was instructed to wear. At the end of the tube was attached a spigot and whenever the pain and distension appeared the spigot was to be opened. This the patient practiced with considerable relief for three months when again the symptoms recurred with their old intensity. It is presumable that the abdomen of this patient has been searched as minutely as practicable for he has been opened by the élite of the profession of both continents, yet he continues to have intense epigastric pain and distension. It would seem that the case is medical and not surgical. It is highly probable that this patient has some abdominal irritation which acts reflexly upon the stomach, but in addition the man is highly neurotic. He has found it necessary to resort to anodynes on many occasions and is in perpetual fear of the appearance of his old enemy. Such a case I believe is to be cured, if cured at all, by the careful study of the organism as a whole, by careful feeding, systematic exercise, the application of the general measures of hydrotherapy and gymnastics together with the all-important psychotherapy. The patient has formed the habit of seeing so-called "distinguished doctors" but though he is so greatly concerned regarding his condition, it is not likely that he can bring himself to submit to the discipline necessary to complete his cure.

Although it is admitted that this case is extreme, I have met with a number of others somewhat similar. These are cases that are not to be explained by spinal cord disease nor by ascertainable lesions in the gastro-intestinal tract. There may be some unknown cause in the abdomen or elsewhere that brings about such unfortunate situations, but to the best of my knowledge the real defect is functional, psychoneuropathic, with no material lesions or at most none that would produce symptoms in an ordinary individual.

The conclusion is that though functional disturbances of the stomach may be produced by irritation in the gall-bladder, cecum, appendix, sigmoid or in the pelvic organs, in which cases these disturbances may be curable by skillful surgical procedures, they may also, even in obstinate and severe cases, be of neuropathic origin. Neither explanation should be too readily accepted. The physical signs of a defective appendix or gall-bladder may be inconspicuous or absent, and some cases, therefore, go without operation that might be thus cured. Such pa-

tients should have the advantage of the united study of physician and surgeon but the physician should have sufficient stamina not to be swayed by the enthusiasm of the surgeon into consenting to operations based upon rather obscure reasons. It is a singular fact that relief is obtained for a few weeks following an abdominal section. An eminent surgeon has described this as a "benefit from the operation per se," but this benefit is temporary and it seems to me that surgeons do not sufficiently appreciate how frequently the old symptoms return in patients who have been dismissed as cured. This is not a place to lay down rules for the differential diagnosis of chronic appendicitis or cholecystitis. It is enough to insist that in cases of functional disturbances of the stomach that arise somewhat abruptly and when after a searching inquiry a suspicious history of symptoms is uncovered that might be referable to the gall-bladder or appendix, these parts should receive the fullest attention and probably surgical intervention.

Case No I. Gastrorrhagia, Dyspepsia, Pyloric Spasm, Jaundice; Operation; Cure.—Mrs. P., 38 years old, had for two years suffered severe dyspeptic symptoms, resembling those produced by hyperchlorhydria. At times the attacks were most acute with intense gastralgia and vomiting. The character of the gastric contents did not explain the symptoms. At the end of one year, accompanying an attack, there appeared a mild jaundice, with sensitiveness over the region of the gall-bladder. Subsequently this region remained tender to pressure, although the pain which recurred frequently, was limited to the epigastrium. After another year of suffering, operation was advised with the idea that there might be cholecystitis. The gall-bladder was found normal, there were no adhesions and no evidence of any other disease.

The appendix, though healthy, was removed and the gall-bladder was drained. To my surprise the patient was cured. Three years have now elapsed without a recurrence of the stomach symptoms.

### THE HEART AND DYSPEPSIA

Heart disease may give rise to dyspepstic symptoms so conspicuous that the underlying cardiac insufficiency attracts little notice. Valvular disease may contribute to this result, but the cause lies chiefly in poor tonicity and contractility of the myocardium. It is not always possible to decide whether this weakness is a result of myocarditis, heart strain, dilatation, or functional depression. Sometimes, we perceive only that the movement of the blood mass is retarded and that the blood pressure is relatively low. More often there is found slight dilatation. One type, which in my experience has been common, belongs to hard working men with large muscular development. Many of these were lumbermen or "loggers" from the Pennsylvania woods. The condition is also found among porters, stevedores and athletes. They complain of gastric distress, eructations. regurgitations, nausea and occasionally of pain. rise in the morning suffering from gastralgia, anorexia There is marked general depression but and dizziness. toward the middle of the day they feel somewhat stronger. are able to take food and then resume work, but at night are in a state of exhaustion.

In some there is depression in gastric secretion, in others a normal gastric chemistry.

In addition to the dyspepsia there is a complaint of lumbar backache. Little acuteness is necessary to discover the underlying circulatory weakness, of which the patient is himself rarely conscious. These cases are cur-

able, yet they require radical measures. Little is gained from symptomatic treatment. It is necessary to obtain prolonged rest in bed. Nauheim baths and medical gymnastics are most useful and so is the taking of nux vomica in large ascending doses, as advised by Musser. There has developed a disproportion between the strength of the cardiac and the skeletal muscles; the aim should be to reëstablish a proper proportion or harmony.

It has seemed to me that the incidence of these cases has shown a relation to epidemics of influenza; however, I am not positive of this. Are there other influences contributing to these symptoms in addition to circulatory depression? Doubtless so; yet I am convinced that this one cause is sufficient.

In functional heart trouble the gastric symptoms are proverbially present. The relationship through the sympathetic (or autonomic) nervous system rarely fails to make itself felt. The stomach disturbances are occasionally important, such as gaseous distension which is at times alarming, gastric atony and gastric unrest with violent eructations. Rarely is the stomach quieted by treating the heart; but the heart is relieved by treating the stomach. The condition is really cured not by treating either organ, but by the upbuilding of the nervous system after having made a careful study of the sources of the nervous derangement.

## DYSPEPSIA FROM IRRITATION OF THE URINARY TRACT

Dyspeptic symptoms of such importance as to mask the actual disease may arise from renal lithiasis, pyelitis, prostatic disease with or without urinary retention, or from deep-seated urethral stricture. The frequency with which this occurs deserves to be emphasized. A good illustration is the following case:

Case No. II. Hyperchlorhydria, Gastric Distress, Uricacidemia, Stone in the Bladder; Lithotrity; Cure.—D. R. was a victim of distressing gastric irritability, with hyperchlorhydria, most rebellious to treatment. The urine was highly acid, but displayed no manifestation of stone in the bladder. Ultimately he had an attack of mild cystitis which led him to consult Dr. Gouley who found and removed by lithotrity a large uric acid calculus, thereby curing the patient of his dyspepsia. Another illustrative case is the following:

Case No. III. Dyspepsia, Distended Bladder; Catheterization; Cure.—An active laborer, for three years had suffered from sciatica with muscular spasm in the legs and from dyspeptic symptoms; marked anorexia, gastric distress, nausea, vomiting, localized tenderness, regurgitation and gas. The gastric secretion was found to be depressed, although not so much as to explain the dyspepsia. During general examination the bladder was found to be distended. The patient was catheterized, the bladder irrigated, and thereupon the vomiting ceased and all other symptoms disappeared with the relief of the urinary disease. It is important to make a routine examination of the genito-urinary tract if we would find the origin of dyspepsia in certain cases.

### **NEURASTHENIA AND DYSPEPSIA**

Not every so-called neurasthenic suffers primarily from a definite nervous or psychic disease, but the experimental studies of Crile would lead us to believe that exhaustion, mental or physical, or shock to mind or body, is attended with transient, persistent or permanent structural changes in the neurones. Not every neurasthenic is troubled with dyspepsia, yet we number it among the rarely absent manifestations. Often the symptoms have a physical basis in the stomach, not causative of neurasthenia, but resultant. Of course, this may be reversed; a structural gastric disease may contribute to neurasthenia. When there is experienced a state of "nervous exhaustion" following a psychic shock, there may quickly develop gastric atony with dilatation, or gastric hyperemia with disturbed secretion and an excessive out-pouring of mucus.

More often a clearly defined change in structure is not ascertainable, yet there are symptoms present in full array. The familiar experience requires little more than mention here as the nervous system in its relation to the stomach is discussed on other pages.

It is well to admit that a proportion of those afflicted with a so-called gastric neurosis are suffering from a functional disorder in a wide sense of the term; they are no more gastric cases than they are brain cases, or sexual cases. This is not invariable, for many times the brunt of symptoms falls on the stomach, just as in others it falls on the heart. The treatment of these requires discrimination. With some the "rest cure" of Weir Mitchell is indispensable; whereas with others regulated, progressive exercise, recreation, the exile of self-pity with the taking of a sensible interest in others comprise the important measures leading to recovery. These recommendations apply to those cases having psychasthenia, dominant ideas and morbid fear, conditions well illustrated in the ensuing.

Case No. IV. Morbid Fear of Drinking Water; Psychotherapy; Recovery.—A machinist, age 35, delicately formed, with an impressionable nervous system, stated that for several years he had been unable to drink water

without serious symptoms, referable at first to the stomach, and later widely distributed. He was conscientious in saying that whether he drank with or between meals the symptoms appeared. If he avoided taking water he escaped the symptoms. He was emaciated, weighing only 107 pounds, although five feet, ten inches in height. For six months, while drinking only milk, he felt no disturbance and weighed 130 pounds. Upon attempting to drink water between meals the symptoms returned and weight steadily decreased. He complained of feeling as if gas were present and of a sinking sensation in the epigastrium, sometimes with eructation. The general symptoms consisted of a feeling of weakness that spread over the body, as he described it, "filling up of the hands and feet." Usually the extremities were slender and shrunken in appearance, but whenever he drank water, he said they plumped out. At the same time there occurred a feeling of cerebral pressure and "swimming." Aside from these symptoms the man felt well and physical examination showed no disease; there was an excellent gastric chemistry and good motion. For the benefit of his health he went west two years ago living on a ranch out-of-doors, leading a wholesome life. His symptoms accompanied him and returned with him, occurring on taking of water. An Ewald test breakfast including the usual amount of water, showed a good digestion and no symptoms developed. He became convinced that his sufferings were imaginary and his health was soon restored.

### DYSPEPSIA IN TUBERCULOSIS

The ability to take nourishment in full quantity, to digest and assimilate it well, is of primary importance in the tuberculous. Usually in the routine treatment of pulmonary phthisis too little attention is given to the

actual condition of the stomach. The dyspepsia of the phthisical often shows itself very early, even before pulmonary changes manifest themselves and in different form; it appears late in the disease, depending then upon an additional set of causes. The dyspeptic symptoms of early phthisis are not sufficiently characteristic to be classed as a separate type. Complaint is usually made of an irritable stomach and of symptoms which conform with those of gastric hyperesthesia. Thus there is distress at varying times after meals, a sensation of soreness or tenderness in the epigastrium with eructation of gas, pyrosis, regurgitation, anorexia and occasionally vomiting. The emesis is sometimes hard to explain, no basis for it being discovered in the gastric contents. As a rule the examinations after test meals show a full acidity, sometimes a hyperacidity, rarely a deficient secre-Some cases show moderate pyloric spasm with delay in emptying of the stomach. When there has been a preceding gastric trouble, it is usually aggravated with the onset of tuberculosis. Undoubtedly any stomach disease which interferes with normal nutrition predisposes to tuberculosis. In the late cases of pulmonary phthisis great variations occur in the actual digestive power of the stomach. Gastric atony causes serious trouble in some, yet real gastrectasis is uncommon. The gastric secretion may be well sustained even in advanced cases, but usually becomes deficient with the advent of high temperature. Riegel believed that, with the reduction of temperature by antipyrin, the gastric secretion was distinctly improved. In the stomach contents there is found the characteristic sputum, particularly in patients who through ignorance or personal peculiarity fail in proper expectoration.

Tuberculous lesions of the stomach, though occasionally

found, are rarely the cause of dyspepsia. This dyspepsia depends upon the nervous irritability and general functional depression which go with the disease, and varies in severity according to the constitution of the patient. In late phthisis must be counted the effects of severe infection, auto-intoxication, anorexia, lowered nutrition, excessive expectoration, fever and hemorrhage, all of which contribute to produce functional depression and hence dyspepsia.

Treatment.—Sufferers from pulmonary phthisis should receive special treatment suited to whatever digestive trouble may be present. Preoccupation as to pulmonary lesions should not lead the attention away from consideration of the gastric side of the case. By so managing the dyspepsia that the patient may eat fully and comfortably much may be done toward cure of the underlying disease. There are no special principles of treatment involved; the tuberculous dyspeptic should be treated as are other stomach cases.

#### INTERNAL SECRETIONS AS RELATED TO DYSPEPSIA

Of diseases connected with disturbances of internal secretion, Graves' disease is one in which gastro-intestinal trouble is common. So marked is this in some instances that W. H. Thompson would account for exophthalmic goiter on the ground of an intestinal auto-intoxication. Without adopting this view, every clinician recognizes the frequency with which the stomach is involved. The symptoms suggest an effort on nature's part to hasten elimination of some toxic substance through the stomach and intestine. At times there is violent diarrhea and intractable vomiting; again there is anorexia and marked gastric irritability. It is possible that the nervous system is merely showing its perturbation here, as else-

where, in disturbed function. However, one can scarcely dismiss the evidence of a chemic, toxic side to these cases.

Gastric disturbance cannot escape attention in well developed Graves' disease, but there is a type in which the symptom complex is incomplete (the "formes frustes" of the French) in which the gastric symptoms so far outstrip the usual symptoms of Graves' disease that the real nature of the case goes unsuspected.

The symptoms are not always the same, but among the more frequent are anorexia, gastric hyperesthesia, vomiting, gastric unrest, aerophagia and hyperchlorhydria. On careful examination there will usually be discovered slight tremor, general nervousness, a suggestion of exophthalmos or other non-gastric manifestations, but these are at times very inconspicuous. The "rest cure" is the proper treatment for these cases, and good results will follow this with patients whose symptoms have resisted the conventional methods of relieving dyspepsia.

The importance of disharmony in, or failure of, other internal secretions than that causing Graves' disease is not so well understood. Yet, that gastric symptoms are produced through disturbances of this order is highly probable. The gastric hormone, described by Edkins, and secretions, such as secretin, are intimately related to digestion. To be considered also are substances which arise at points remote from the stomach and concerning which our knowledge is inadequate. Certain gastric symptoms of pregnancy depend upon causes belonging somewhere in this realm. The dyspepsia attending the menopause seems to be related to the change in, or disappearance of, the ovarian internal secretion, and sometimes is relieved by the administration of the extract of corpus luteum. A corresponding relationship seems to

exist with the orchitic internal secretion in the male, and though the subject is in many ways a difficult one, competent observers are convinced of the general truth of the contention of Brown-Sequard. It is undoubted that the internal secretions are to be considered as important factors in digestion, producing by their derangement marked gastric symptoms.

### DIATHESIS AND DYSPEPSIA

Hereditary metabolic peculiarities, the expression of which is recognized in diathesis, produce symptoms depending in degree upon the incompatibility of the routine habits of life of the individual with the special needs of his organism as conditioned by the diathesis. early life when the nutritive processes are vigorous, the possessor of a gouty diathesis, provided he leads an active, outdoor existence, may be free from symptoms regardless of diet. When he follows a sedentary occupation and lives indoors, the troublesome manifestations of the diathesis make themselves felt unless the diet is properly selected and restricted. At middle life and later, the metabolic activity is decreased and, in proportion to this, exercise must be taken and diet adapted to the enfeebled powers of oxygenation. Such individuals have appetite in excess of the needs of the organism which protects itself from the evils of super-alimentation through disturbances of the primae viae without which there would be greater development of the condition known as lithemia. Under these circumstances it is common to find an irritable stomach, hyperchlorhydria, a sensitive pylorus, gastric unrest, etc. Various hepatic disturbances are likely to supervene. Many chronic dyspeptics are in fact suffering from latent gout, and are best relieved by treatment which takes this into consideration and which lessens the intake of purins and promotes their elimination.

Closely allied to latent gout, or lithemia, is that diathesis in which oxaluria occurs as a prominent manifestation. This condition of oxaluria, concerning which Golding-Bird and Prout wrote wisely more than half a century ago, has been much neglected by clinicians in recent years.

All students of the question refer to the very frequent association of dyspepsia with oxaluria. Dr. Deboutd'Estrées of Contrexeville, whose extensive experience with lithiasis and allied disorders compels attention, believes that catarrhal gastritis and dyspepsia contribute to the development of oxaluria. He has noticed that formerly oxaluria was rare in Europeans, but frequent in Americans, though he found the reverse true of uricacidemia. He says: "While the French, sedentary by taste or by profession, give themselves up too willingly to high living, the Americans live too fast, fatigue their nervous systems beyond reason, without giving their digestive apparatus that proper care and attention which it deserves." Of late he has observed the appearance of oxaluria in increasing proportion among Europeans and attributes this to the same causes, namely, "to overwork and fatigue of the nervous system as we (the French) become more and more like Americans." Of course, it is understood that oxaluria is readily produced by overindulgence in foods rich in oxalic acid, such as rhubarb, tomatoes, celery, chocolate, etc., but the victim of this diathesis is subject to oxaluria even when subsisting upon an ordinary diet and is not exempt save when the diet is selectively restricted and when open-air exercise is actively followed.

The conclusion that dyspepsia with imperfect primary

digestion predisposes to oxaluria is doubtless correct, yet I am satisfied that the state giving rise to oxaluria is to some extent hereditary; that it is closely related to, and often interchangeable with, the gouty diathesis, and that not only is it produced by faulty digestion, but that it of itself sets up dyspepsia that will persist in spite of treatment, until the diathesis receives adequate attention in the plan of therapeutics.

It seems probable that persistent oxaluria, as noted in some persons throughout many years in life, is in some way related to a disturbed calcium metabolism.

M. Loeper of Paris, who has carefully studied oxalemia, describes what he terms oxalemic gout in which the stomach is more often attacked than in classical gout. Usually there is an irritative dyspepsia, sometimes an actual gastritis, both of which appear to depend upon irritation by the presence of crystals of calcium oxalate in the gastric mucosa, which have found their way into the stomach through an attempt at elimination. Loeper found these crystals both in the gastric mucosa and in the stomach contents. The intestine suffers in a similar way, and this seems to be analogous to what occurs in the kidney in cases of oxaluria.

The symptoms most often present in oxalemic dyspepsia are a sense of burning, irritation, eructation, waterbrash, and at times vomiting. Pyloric spasm with pain and motor insufficiency may occur and, in rare instances, hemorrhage, small in amount, but accompanied by considerable distress.

Loeper announces that there is present in the blood of these cases numerous calcium oxalate crystals, and he states that the gastric mucosa, in experimental cases, shows punctate hemorrhages, erosions and other changes. The condition is not always associated with oxaluria, for often during the visceral attacks the oxalates, habitually present in the urine of such patients, temporarily disappear.

Treatment.—The diet should be such as to limit the intake of oxalic acid and the purin bodies. It should, therefore, be abundant in rice, fine flour bread, milk and the various milk foods. Alkaline waters containing the more soluble bicarbonates should be freely prescribed. Also, the patient should lead a life of physical activity in the open air. This exercise should be regulated so as to avoid over-fatigue which might influence the nervous system or the circulation adversely. With such treatment this form of dyspepsia will disappear though it may have resisted conventional treatment.

The value of the alkaline treatment above-mentioned attracted my attention when I found unexpected improvement in a case of protracted dyspepsia after the administration of an alkaline mixture which was prescribed for the relief of an over-acid urine and visceral irritability.

```
      Potassii nitratis
      .50 (gr. viii)

      Sodii bicarbonatis
      1.00 (gr. xv)

      Sodii et potassii tartratis
      2.00 (gr. xxxii)

      Sig.—Take, dissolved in a glass of water.
```

An important principle of therapeutics I learned from Debout-d'Estrées. It is this: when there is infection, for instance, of the digestive or biliary tract, or of the genitourinary apparatus, it is difficult to raise the specific immunity of the organism so as to overcome the infection, until the faults of the diathesis (the latent gout, the oxaluria) have been subdued. When a general nutritional balance has been obtained, the infection and its consequences become of less importance and may be successfully dealt with if they do not disappear spon-

taneously. By applying this principle to dyspepsia, much may be gained in the understanding of the pathogenicity and of the therapeutics of many cases.

Such a diet and such a régime in general lead to the disappearance of oxaluria and oxalemia and dissipate the symptoms, prominent among which are gastric disturbances and closely associated functional hepatic disturbances. The treatment which overcomes dyspepsia in these cases of oxaluria and gouty diathesis is also the treatment which is necessary, if subsequent high blood pressure, arterio-sclerosis, renal, cardiac and other structural changes are to be avoided.

# DISTURBANCES OF THE SPECIAL SENSES IN RELATION TO DYSPEPSIA

It is remarkable how steadily but strikingly digestion may be distributed as a result of an offense of taste or smell. The functional derangements produced by a disturbed sense of smell have received but little attention. Wende and Busch 1 of Buffalo have reported a group of cases in which a peculiar sweat reflex was excited by the odor arising from certain articles of food, noticeably, tomatoes, strong cheese, roast beef and pickles, and in one case, onions. Although the symptom was sometimes observed while eating, nevertheless it was shown that the sweat reflex depended on a disturbance in the sense of smell. Heredity was a feature in all these cases. It is well known that asthma may be induced in this manner, a manifestation which is analogous to the marked disturbances of gastric function which are induced through sensory impulses.

Motor insufficiency and gastric distress not infrequently accompany sinusitis with defective drainage

<sup>1</sup> Jour. Am. Med. Assoc., July 17, 1909.

from the frontal, ethmoidal, or antral chambers, and similar results may accompany mastoiditis.

In the disturbance of the organs of special sense, as in appendicitis, cholecystitis, etc., the presence of special autonomic or sympathetic nerve irritation must be considered; also the presence of the vagotonic or sympatheticotonic state of the individual. This refers to eye-strain in particular.

Abnormalities of the taste sense have an important bearing upon the functions of the stomach. Such disturbances may be a consequence of central nervous disease, which explains the fact that they sometimes occur in hemiplegia and hemianesthesia. Disturbance of the sense of taste may accompany neuritis. There is some imperfectly understood relation between disease of the middle ear and the sense of taste. In neurasthenia and hysteria these taste perversions sometimes assume great importance, not only because of the resulting anorexia, but also because of interference with the gastric digestion. Disease of the buccal mucous membrane or glossitis may lead to depression and to other derangements of the sense of taste. It is a well-known fact that from the presence in the blood of certain substances which reach the gustatory organs through the circulation, there may result very marked disturbance of taste.

Unpleasant sensations of hearing and sight are to considerable extent overlooked through training, yet consciously or unconsciously to us these may affect digestion unfavorably. The relation between the special senses and the digestive apparatus possesses more importance than is usually appreciated.

Eye-strain and Dyspepsia.—There have been so many acrimonious discussions on the relation of eye-strain to dyspepsia that the profession has become impatient of

the subject. However, one finds that a calm examination of the facts and a careful application of the methods of diagnosis by exclusion, are the exception rather than the rule. Able clinicians arbitrarily explain the good results which follow the careful correction of refractive errors to a changed mental attitude, the result of suggestion. In few questions of pathological cause of symptoms is there so much intolerance. Many oculists attribute to eyestrain practically all functional disturbances including gastric disorders. The deliberate turning aside from the question, as well as the magnification of one or another. of its aspects, is resulting in confusion and, in no little needless suffering on the part of patients. For instance, we meet with those who suffer, occasionally with life jeopardized, from an unrecognized appendicitis, and who are led to believe that their trouble is solely the result of a mysterious pair of eyes which can be relieved only by often repeated correction of refraction or by the shortening or lengthening of certain eye muscles. On the other hand we find dyspeptics having persistent symptoms that later are shown to depend upon an uncorrected astigmatism, yet who have submitted to unnecessary restriction in diet and harmful medication, to appendectomy and to uterine fixation.

The truth is that eye-strain unaccompanied by any known structural disease is of itself sufficient to produce aggravated dyspepsia and other functional disturbances. Most physicians recognize this fact and although they refuse to discuss the question they nevertheless refer their patients to oculists and often with most satisfactory results. Nevertheless the question is a very embarrassing one and is not to be settled by the method of exclusion as readily as is the case with most definite diseases. This depends upon the following facts.

- (1) There are many individuals who have striking defects of vision who escape from the various functional disturbances which eye-strain may and does produce in others. I do not know how this is to be explained. It may depend upon the readiness with which in some cases Nature adapts herself to a fixed strain and abnormality; whereas in other cases, where greater susceptibility exists, she fails in this adaptation. This may seem to be but the expression of the reasoning of old medicine rather than induction from facts, but it remains true that countless carefully recorded cases show wide variation in adaptation to ocular defects.
- (2) Another important difficulty is that more than ordinary skill is necessary for the discovery and correction of certain ocular defects, and this is true precisely in that class of cases most susceptible to eye-strain. One would suppose the correction of refractive errors to be an exact science, based upon measurements that could be made by any well trained and careful oculist. Experience does not justify this belief. I have referred patients to five different oculists in succession at short intervals, with the identical request to each that he make a complete examination of the eyes and submit to me a written report of both the result of the examination and the correction which should be prescribed; and, strange to relate, I have received a different report and a different prescription from each of the five oculists. More than once I have repeated this experiment, not always referring to five different oculists, but several times to three in succession. This is a hardship to the patient and would have been an offense to my conscience but that I felt sure that the patient's symptoms depended upon eye-strain, and experience showed that those cases in which were seen these remarkable discrepancies in the

diagnosis of the oculists were cases in which, ultimately, striking benefit resulted from the correction that suited. It would be unjust and discreditable to attribute these varying results to the lack of conscientiousness or training on the part of the oculists in question. All were competent men and each was desirous of cooperating in discovering the source of the functional symptoms of which the respective patients complained. There seems to be a difference in the mental attitude with which different oculists approach these cases. The matter would be more easily disposed of were a certainty of result the invariable outcome of the work of one man or of certain men; but that is not the case, and where one man fails another succeeds, and vice versa. My meaning is that even a competent man, successful in many cases where others failed, may be unsuccessful on some particular occasion, while his neighbor, competent but no more so than he, may have a brilliant result. Some oculists recognize this fact and cordially cooperate in the attempt to reach a conclusion on an obstinate refractive case. greatly indebted to the often disinterested efforts of these gentlemen in relieving dyspeptics having aggravated and long standing symptoms.

(3) There is another reason for misunderstanding the part played by eye-strain in causing dyspepsia, and this is that often there are several detached yet related factors which contribute to produce the complex in a given case, and the symptoms persist until all these factors are removed, although a moiety of relief may be obtained by overcoming one of the sources of trouble. For instance, a patient may work in an unsanitary room and, from imperfect ventilation, metabolic disturbances may develop and from the attending toxemia the stomach may be irritated. The matter is made worse by cecal stasis and

constipation, and finally the symptoms may overflow his measure of endurance if he loses his eyeglasses and therefore adds eye-strain to the other contributing causes of his malady. It requires no little openness of mind and self-examination on the part of the clinician, not to allow some of these causes to escape his observation. Human frailty seems to show itself in a satisfaction at finding a singleness of cause, to the extent that when the mind is turned to perceive one side of a problem it becomes oblivious of the other.

In order to be successful in the treatment of dyspepsia one must be alert to many things. The inheritance of the patient, the past history and home life must be understood. The personal habits must be learned, the diet. the meal hours and manner of eating. We must recognize the fact that some special and probably unsuspected kind of food may be noxious; we must exclude local stomach trouble, such as gastritis or ulcer, and we must weigh the possibility of reflex disturbance, such as comes from appendicitis, or an uncorrected eve-strain. When the subject of dyspepsia is open-mindedly studied, when the physician is not too incredulous as to possible exciting causes and when he is able to square his conclusions with results of careful analyses of stomach contents, the best opportunity for successful diagnosis and treatment is afforded. One of the most frequent occasions of dyspeptic symptoms is found in change of occupation. A man who has habitually worked in the open air and has accustomed himself to eat heartily of relatively coarse foods, is likely to develop dyspepsia when he undertakes work that requires close application of the eyes and a sedentary, indoor life. The dyspensia might have been avoided had the dietary been adjusted to the changed condition. An illustration may be found at a family table. The father is a laborer in the open air, the mother busy with household occupations and the daughter a school teacher. Now under such circumstances some one is sure to suffer. If the food is sufficiently coarse and hearty to satisfy the father it will be found to be indigestible in the case of the daughter and unappetizing in that of the mother; whereas if the diet is changed to conform with the needs of mother and daughter it will prove insufficient and unsatisfying to the outdoor laborer.

## GASTRITIS AND FUNCTIONAL DYSPEPSIA

During the operation of any of the various conditions that consume the vital energy of an individual, as for instance, insomnia, overwork, unhygienic surroundings, eve-strain, insufficient or improper food, it is easy and natural to have ensuing gastritis added as a cause of dyspepsia. We have previously noted that there are many causes which by depressing the energy and making the nervous system irritable, lead to functional disturbances, which may so harass the economy that slight eyestrain becomes important. Let us now recall that it is under these same depressing influences that immunity to infection is lost; and with a decrease in immunity, with the lowering of resistance on the part of the stomach to the invasion of microörganisms, there may be a localized or generalized gastritis. This probably explains, in part at least, the conflict between those who hold that functional dyspepsia is the expression of an inflammatory process and those who hold that it is based upon no structural changes whatever.

## THE NATURE OF DYSPEPSIA

Dyspepsia, therefore, is not any one thing. It is often a sympathetic disturbance and may be produced by a

great variety of conditions bringing about gastric distress. It does not necessarily mean indigestion or overdigestion, hyperchlorhydria or anacidity, excessive motor activity or gastric atony. It does not necessarily mean food intoxication nor derangements in vital chemistry; nor appendicitis, cholecystitis nor eye-strain; finally, it does not of necessity mean gastritis. sia does mean a break in harmony in the complicated physiology of the digestive apparatus. The break in physiologic harmony may be induced from any one, or from the combined effect of several of the various exciting factors which have just been listed, or others not here included, for of course the catalog could be enlarged. This disruption of physiologic harmony, which we call dyspensia, manifests itself in a comparatively small number of symptoms, such as pain, distress, hunger, anorexia, eructation, regurgitation and distension. But the derangements of physiology which give rise to these symptoms are numerous, and there may be different physiologic disturbances in different individuals from common cause. That is to say, that which leads to hyperacidity in one may bring about lowered secretion in another; in a third, atony may develop. Numerous are the combinations of disturbed physiology that are easily exposed in the study of dyspepsia, and he would be an ingenious man who could insure that a particular physiologic abnormality invariably resulted from any one cause. It is as if numberless fibers of different textures were united to form a common coil which, when unraveled, might be woven into many fabrics of dissimilar character.

# TREATMENT OF DYSPEPSIA

If we are to relieve the dyspeptic we must divorce ourselves from set formulae and from conventional diet lists. The stomach is protean in its complaints and its desires, and the vagaries of the dyspeptic at times seem to us most fanciful. Indeed there is here ground for easy self-deception on the part of the patient, and auto-suggestion displays many peculiarities. Doubtless we are at times unjust in denying the conclusions of the patient. On the other hand, nowhere is a patient more likely to be self-deceived than in judging the disturbances of sensation related to his dyspepsia. These statements may seem inconsistent with the somewhat dogmatic measures of treatment about to be set forth.

It goes without saying that the best way to cure a dyspeptic is to remove the source of the trouble. Therefore. the plan of treatment should not be undertaken until after a searching general examination has been made. Then we should remove as far as we can the various exciting causes and proceed in our attempt to bring about greater harmony in physiological activity. It is of primary importance to discover and apply the just means of restoring the patient to a greater degree of general physiologic power. It would seem as if there were a vitality-containing receptacle the pressure upon which varies. When this pressure is at a proper height, physiologic activity moves on smoothly giving no occasion for complaint. When this pressure is too low, functional symptoms abound. Therefore we must improve the patient's vitality. By some means we must get him into a state which will permit of slight temporary excesses of one kind or another without so lowering this vital pressure as to develop symptoms. Experience teaches that this often can be done. We can raise the general energy of the patient if we keep that side of the problem steadily before us. The exceptions with which we are familiar are those patients who are suffering from tuberculosis or other disease which lowers energy more rapidly than we can restore it. None the less, this problem remains the same and we must restore energy as the best means of procuring a harmonious working of the machine. Very often the most needed thing and that most difficult to obtain is rest. This sometimes means rest in the fullest sense of the word and sometimes rest of a special apparatus. It may mean rest of the mind and exercise of the body or perhaps rest of the body with proper recreative use of the mind; which reminds me of O. Henry's story in which the physician made to his patient the assertion that "what he needed was exercise and rest." Fortunately for us, unless struggling with some wasting disease, nature will restore lost energy if given an opportunity. That we have learned this is shown by our continual demand that our patients change their way of living. If the organism has become exhausted under one course of life, it seems probable that restoration may result from a change. We must be discriminating and far-sighted in finding the leak through which energy is being wasted. With energy restored what more is there to do? In most cases, there is nothing more to do, for the patient finds himself well. But will he remain well? Probably not unless we devote ourselves to educating him concerning the cause and effect in the matter of dyspepsia. tendency with the majority of these patients is to follow the line of least resistance and to fall back into the old routine that led to a lowering of resisting power, whereupon there is a recurrence of functional excitability and probably a re-awakening of gastric over-consciousness. Therefore we should educate our patients in the method of proper living. With the same painstaking care which we exhibit in laying out the course to be followed by the

tuberculous patient, and following somewhat the same course, we should instruct our patients what to eat, how to eat, where to sleep, how much to exercise and how much to rest. I have spoken of the resemblance between the plan for the dyspeptic and that for the tuberculous patient. On reflection we shall see that the rules of life that best suit the tuberculous are those which raise to the highest plane the general nutrition, the powers of assimilation and that which is an integral part thereof, the primary digestion. In neither tuberculosis nor dyspepsia does this mean invariably the practice of super-alimentation and rest. In both cases super-alimentation and rest are adopted as the best means of raising the patient's resisting powers; yet every clinician knows that there are dangers of overfeeding the tuberculous patient and also that there comes a time when exercise is absolutely necessary. To some, it will seem absurd to speak of full feeding as a measure suitable for the dyspeptic. Such a conclusion would be hasty. In about 50 per cent of dyspertics there is need of super-alimentation. They are suffering from starvation, which induces nervous sensitiveness and organic erythism. However, it is necessary even in these cases for us to understand the peculiarities of metabolism in each case. In a given instance we may allow the animal proteids in abundance with a corresponding decrease in carbohydrates, yet this practice if followed for too long a time may, especially with that numerous group who are heirs to gouty tendencies, perhaps latent, induce the condition which we call lithemia. The lithemia probably will give rise to hepatic congestion and thus to a recurrence of dyspeptic symptoms. Lithemia is not an identical state in all cases in which we apply the term. No precise description of its deviation from the normal vital chemistry can be

given. In a general sense we know that these patients have over-acid urine and are benefited by the taking of alkalies. Some of them are victims of oxaluria, some of phosphaturia, and there may be abrupt and unaccountable oscillations between urinary alkalinity and over-acidity. Even though we are not sufficiently acquainted with the metabolic fault in every instance, we at least know that the derangement may be corrected most satisfactorily by diet suiting to the individual case. This brings us to the consideration of caloric food values and the number of calories or food units that should be supplied to a given individual, a subject which at this time is attracting considerable attention. There is no practical method by which we can reach an exact knowledge on this question and generally we must be content with the empirical method of finding the diet and the number of food calories which the case requires. It is highly probable that we should do better by requiring more cases to go to the inconvenience and expense of having a careful physiological study of metabolism. This course we find necessary in cases of interstitial nephritis, hepatitis, diabetes mellitus, etc. The empirical method, although useful, may be greatly assisted by that information which the physiological laboratory alone can give us. portance of having the benefit of these physiological studies is recognized in cases of diabetes and chronic nephritis, but after all is that not like "locking the stable door after the horse is stolen"? Would it not be better to anticipate the serious organic changes which express themselves in the symptoms of auto-intoxication? Unquestionably a better knowledge of metabolism, in those who are known to be lithemic and who suffer as a result from dyspeptic symptoms, might enable us to avoid an oncoming metabolic crisis and to avert a serious

organic disease, which otherwise occurs as a legitimate inheritance. The method of "tracking" disease is most It is comparatively easy to retrace the steps profitable. which a disease process has followed, but it is more difficult to foresee the path that it may take. Yet we know that we gain in the power of doing this through making careful observations, recording them and thinking. I am led to these reflections by recalling the "health curve" of patients whose career I have watched for years. thoughtless and improper diet, the insufficient and illtimed period of exercise, the continued nerve strain, the developing dyspeptic symptoms, the insufficient alimentation, the overtaxed and broken metabolism, the lithemic symptoms, the hepatic and renal insufficiency, the final arterio-sclerosis and nephritis; these are the mile posts that I have seen along the road.

I hope that the reader may forgive this seeming discursion; possibly some may regard it as the writing of platitudes. I realize that I am stating facts well known, but apparently the facts are too often disregarded or they are not arranged in such a manner as to apply directly to digestive diseases.

It has seemed to me that correlation of these well known facts is too often neglected, and that we permit ourselves to overlook the meaning of the pathologic indications which in the beginning are so very often those of digestive abnormality. It is for the reason that I have seen what appeared to be a disregard of the relationship in every day pathology as expressed in disturbances of digestion that I have felt a desire to write this book. If the lesson which I am attempting to teach is erroneous or unnecessary then the work has no raison d'être. But believing the contrary, especial stress is given to those digestive affections which, through iden-

tity in etiology and through association in their manifestations with other better defined and more immediately disastrous diseases, are misunderstood or disregarded and their significance lost. Is it not remarkable that so often the chronic infections, the intoxications that lead to or accompany most important pathological processes, trumpet their arrival with the symptoms of dyspepsia? Doubtless the chief reason that we overlook the meaning of this is because these same symptoms so commonly announce merely an irritation that is comparatively trivial in character; however it should serve to warn us that these stomach symptoms are not to be put aside without thoughtful investigation, and it suggests that sometimes by the successful use of palliative measures we may cover the evidences of oncoming disease until the time arrives when we are unable to successfully combat them. It is for this reason that the general practitioner has felt an instinctive suspicion of relinquishing to specialists the care of dyspeptic cases. There is some ground for this view, but it should not be allowed to prevent taking advantage of the progress in the knowledge of the digestive diseases which has been made through the work of the indefatigable specialist. The diseases of digestion can not and should not be relegated to a specialty, but general medicine is in need of investigation which can only come from special training.

To return to the more definite question of treatment, it will be found that after removing the cause of a digestive disturbance, after having raised the resisting power of the patient, and after having given him directions as to a proper course of living, that there still remains to be considered the treatment of certain special conditions which depend at times upon individual peculiarities or defects, the nature of which may not be de-

finable. We recognize their presence and while we do not forget the importance of the general method of treatment already considered, we find it necessary to give attention to these individual tendencies which result in producing such definite disturbances of the physiology that we have been tempted to regard them as distinct neuroses. Some of these conditions undoubtedly can be properly so classified, others do not fall under the head of the neuroses, but for convenience may be so grouped. They are merely the common derangements of digestion that are excited by the general causes which have been discussed. However, it is well to give them individual consideration, yet always with the understanding that they are not to be regarded as clinical entities but as instances of physiologic disturbances, tending to persist along fairly definite lines and sufficient to convince the patient that he is dyspeptic.

Before entering upon the consideration of these special functional disturbances of digestion, it may be well to repeat that we must constantly be on guard lest the symptoms have a definite physical basis in a local disease of the stomach. Always when dealing with these functional disturbances it is wise to suspect the presence of a local cause. Therefore, while dealing with one condition, we must be searching for the other, and thus we may discover incipient cancer, ulcer or gastritis. Gastritis especially, plants itself in the soil made ready by functional disturbances, and as shown in another section, it is often taken to be dyspepsia of the neurasthenic.

## CHAPTER IX

FUNCTIONAL DERANGEMENTS OF DIGESTION AND GASTRIC NEUROSES

As before stated, the term "dyspepsia" has fallen somewhat into discredit. When the disturbed physiology of digestion came to be better understood, when the matter of over- or under-secretion, excessive or depressed motion, etc., became recognized, these special conditions came to be considered entities and treated as such. have come to appreciate that there does not exist that wide difference in nature between hyperchlorhydria and hypochlorhydria or between gastric unrest and gastric atony as was first believed. We understand now that though the manifestations of these various functional disorders are quite dissimilar, their causes are closely related. That is to say, similar etiological conditions lead to disturbance of gastric physiology, and although the disturbances are not identical in all cases, they are nevertheless based upon similar causes and are therefore more or less closely related. In one patient we may have developing an excitement of the stomach in its various activities, in another a depression, and in still other cases we may have excitement followed by depression. convenient and sometimes necessary to speak of a case as one of hyperchlorhydria, or hypochlorhydria, or excessive peristalsis, or atony. From a therapeutic and dietetic standpoint it is important to recognize which one of these various disturbances of gastric physiology is However, I believe a misconception arises dominant.

from the statement that any functional disturbance, such as hyperchlorhydria, hypochlorhydria, etc., is a distinct entity, unrelated to other functional derangements for they are closely related and in individual cases we from time to time find several somewhat definite physiological disturbances occurring one after the other, or more commonly in groups. These groups of disturbances exist in sufficiently distinct association with each other to make possible a division of the functional disturbances of the stomach into types of functional diseases. These types follow, as a usual rule, paths that may be foreseen and predicted so that accidents of the case may be anticipated.

Symptoms and Signs.—The symptoms and signs of the different types of functional disorders of the stomach are not so unlike as has been stated in some text books. There are cases in which from the symptoms and signs alone it would be impossible to determine whether the case belonged to one type or another. The truth can only be elicited by repeated observations and repeated gastric analyses. For this reason the old term "dyspepsia" seems better to me than the term "indigestion" which displaced it. In fact it is often misleading to say that there is indigestion, for at times the digestive activity is exaggerated. Whether digestion is overactive or much retarded, it occasions uneasiness and distress, that is. "dyspepsia."

Vagotonic and Sympatheticotonic States.—Those types of dyspepsia in which there has occurred an excitement or an exaggeration of function are now by certain physiologists attributed to over-action of the autonomic nervous system and are spoken of as vagotonic states. On the other hand, those dyspeptic conditions attended with lowered motility and secretion are regarded as resulting

from an exaggeration of influence on the part of the sympathetic nervous system and are spoken of as sympatheticotonic states. These views change but little our conception of the nature of functional gastric disturbances. The innervation of the digestive organ is complicated, and it is probable that the assertions now positively made as to the bearing of vagotonic and sympatheticotonic conditions in explanation of gastric disturbances eventually will have to be modified. It is convenient to speak of over-tonicity, over-contractility and over-secretion as vagotonic, and of functional atony, motor insufficiency and lowered secretion as sympatheticotonic states. In using these terms, however, it should be understood that some reservations are made in certain instances.

# TYPES OF FUNCTIONAL DISTURBANCES ANOMALIES OF SECRETION

In part the gastric juice seems to be secreted in response to an internal secretion or hormone arising in the gastric mucosa. In some instances there is a high acidity without a great amount of free HCl, although the ferments may occur in large amount. In these cases the total acidity depends for the most part upon the presence of an excess of combined chlorids. This condition gives rise to fewer disagreeable symptoms than attend true hyperchlorhydria. In other cases we meet with excessive secretion of thin watery mucus which may have its origin in an obscure inflammatory condition. There are still other cases in which secretion of mucus in the stomach seems to be below normal, and under these circumstances the patient complains of irritability of the stomach with symptoms that suggest hyperesthesia as mentioned by Kaufmann.

# HYPERCHLORHYDRIA

When the total acidity of the gastric contents continues at 60 or above, it is commonly spoken of as hyperacidity. When, as is commonly the case, this hyperacidity depends upon the presence of free HCl, and combined chlorids, the condition is called hyperchlorhydria.

In the beginning we must recognize that it is a fallacy to consider all cases having an acidity above 60, depending upon HCl, to be instances of hyperchlorhydria. There are wide individual differences, and relatively high acidity may be normal to one person and a relatively low one normal to another. There seems to be no satisfactory way of explaining this fact. The average individual in town life has, fifty minutes after an Ewald breakfast, an acidity of about 50. When this acidity increases to 60 and up to 100, it is frequently accompanied by definite symptoms which have come to be attributed entirely to the high acidity. But even an extremely high acidity does not always produce these symptoms. some extent their appearance or non-appearance depends upon the degree of general resisting power of the individual. A hearty, unimpressionable, muscularly active man who lives much in the open air and who may subject himself without suffering to marked changes of temperature and other physiological strains, will often be found habitually to carry a total acidity of 70 to 100 or even more without the development of symptoms usually attributed to hyperchlorhydria. On the other hand, in the case of a delicate woman of sedentary habits, whose organism is so poised that moderate physical strain produces fatigue or some variety of distress and who may normally carry an acidity of only 25 to 30, symptoms may appear at once if, from any cause, this

acidity rises to 50 or 60, the so-called normal line of the average individual. Let it be understood then that hyperchlorhydria cannot be positively measured in terms of acidity of the gastric contents. In other words, the exact degree of acidity is merely an approximate value; and the diagnosis must rest not alone upon the acidity of the gastric contents, but upon the results produced by this acidity on the patient. This matter seems to be very simple, yet it is one on which physicians are constantly going astray. With some the tendency is to allow too much importance to the mere question of gastric acidity. others disregard the state of the gastric contents and erroneously attribute the symptoms to some local lesion of the stomach, such as ulcer, erosion or gastritis, or to food stagnation or possibly to systemic or reflex conditions. There is more or less truth in each of these views, but the important thing is to decide to just what degree one or several of these influences may be at work in the case on hand.

Etiology.—There is no one cause for this type of gastric neurosis. As I have said before, causes that in one case are responsible for an over-excitement of secretion in another case may produce lowered secretion, or still in a third case disturbances of motion or sensation. general it may be said that any cause that lowers physiological resistance is competent to produce these effects. Records of cases show that the condition is often secondary to over-work, especially over-taxing of the nervous system, loss of sleep, worry, grief, eye-strain, lowered nutrition, etc., also to reflex nervous causes, such as stone in the bladder, retroversion of the uterus, sigmoiditis, stagnation in or irritation of the cecum, appendix gall-bladder, etc. It is so common a result of cholecystitis as to mislead some into concluding that usually

cases of hyperchlorhydria, when not dependent upon ulcer are dependent upon disease of the gall-bladder. This conception of the condition is incomplete. The physician must be on the alert not to overlook local disease in the neighborhood of the pylorus while attempting to explain the symptoms by hyperchlorhydria of unknown origin. One should always search eagerly for the cause of a functional derangement of digestion. It sometimes requires a wide knowledge of medicine and careful differentiation to uncover this exciting cause. It is probable that hyperchlorhydria may be due to abnormal excitement of the gastric hormone described by Edkins, although I am unaware of any experimental demonstration of this.

There is experimental proof that hyperchlorhydria and over-secretion may be induced by stimulation of the vagus nerves; therefore it may be permitted to speak of these conditions as vagotonic.

Symptoms and Signs.—The appetite is usually good in hyperchlorhydria. An hour or two after eating the patient complains of gastric uneasiness and a burning sensation and sometimes pain in the epigastric region. There is generally a disturbance of gastric motility which evidences itself in eructations of gas, regurgitations of sour fluid or water brash, and very rarely in vomiting of sour fluid. When the acidity is very high there is apt to be over-tonicity or even moderate pyloric spasm, and gastric juice remains too long in the stomach. All these symptoms are likely to be aggravated by an over-stimulating diet. This includes condiments, acids, coarse foods, and those foods that offer great resistance to digestion, for instance warm pastry, fresh bread, iced, or highly seasoned articles of food. Sometimes the symptoms appear after a small meal which stimulates more

secretion than can be utilized, sometimes after large meals, because there is insufficient energy on the part of the stomach to make the required effort. After these symptoms have continued for some days there occurs constipation, slight congestion of the liver and concentrated urine. There is usually considerable general nervous perturbation. Careful palpation over the stomach may reveal the presence of too much fluid and there may be a diffuse general tenderness of the epigastrium or unusual resistance upon palpation, the result of over-tonicity. Neither the tenderness nor the distress is so localized as in ulcer. The symptoms usually develop somewhat later after eating, as is true also in gastric ulcer. Often the pain first occurs as late as two hours after a meal. a symptom which is also commonly present in duodenal or pyloric ulcer.

Loeper 1 describes the action of an internal secretion, which is derived as an alcoholic extract from the fundus of the stomach especially, and which differs from the gastric hormone of Edkins in that it is much enfeebled by heating. To it he attributes the source of pensinuria. The presence of a digestive ferment in the urine is, he finds, most noticeable in hyperchlorhydria, especially with benign pyloric stenosis. It is with these same conditions that this internal secretion is most active. It produces a noticeable depression of vascular tension, occurring immediately after a meal, especially of meat; then there is a rise, followed by a second depression of vascular tonus. It is a fact worthy of consideration, that patients with hyperchlorhydria, especially with pyloric stenosis, as a rule have relative hypotonia. Exceptions, of course, occur with advanced arteriosclerosis, yet in other instances, even then there will be found a lower

<sup>&</sup>lt;sup>1</sup> Sem. Méd., 3 mai, 1911.

pressure than would be expected. In reviewing a series of cases taken in order with a gastric acidity of 70 or over, not more than one in ten had a blood pressure above 140, and an equal number had a systolic pressure at about 100. There are other possible reasons in these cases for low blood pressure besides the effect of an internal secretion, yet it is an interesting question as to how much the vascular as well as other symptoms of hyperchlorhydria may depend upon an exaggeration of internal secretions.

Diagnosis and Differential Diagnosis.—In the diagnosis of hyperchlorhydria we should consider: (1) the group of symptoms associated with the peculiarities of the stomach contents. These are discomfort or pain in the stomach occurring, as a rule some time after meals, with eructation, water brash and slight diffuse gastric tenderness. Usually the appetite is not impaired; rarely there is acid vomiting. The symptoms are relieved promptly but temporarily by the taking of food. After an Ewald test breakfast, the stomach contents show a high acidity, with poor digestion of starch. Digestion of proteids is rapid as may be found by giving a test meal of minced meat, bread and water. Conclusion as to the true character of the gastric secretion should be made only after repeated examinations.

(2) We should consider the soil on which this symptom complex has been developed; that is, the temperament of the patient and the lowered nervous resistance from general causes. Also, we should search for any active local cause which may reflexly produce general gastric excitement, and hence over-secretion. It is not difficult to recognize hyperchlorhydria, but it may be difficult to exclude a definite local or reflex cause. In order to exclude special causes for hyperchlorhydria, we should

search for conditions to which it is ordinarily secondary. In peptic ulcer hyperchlorhydria is the rule, yet it is not invariable. The stomach contents in ulcer almost always shows red blood cells or occult blood on careful and repeated examinations. Sometimes blood-stained wash water may be recovered. The pain in ulcer is localized and is apt to appear soon after eating, except when the ulcer is at the pylorus or in the duodenum. tenderness over the seat of pain, and referred tenderness is felt just to the left of the spine between the tenth and the twelfth ribs. Nausea, vomiting and hematemesis of gastric ulcer should be considered. Duodenal ulcer is more difficult to exclude. The character of the pain is sometimes identical with that of simple hyperchlorhydria. There is usually tenderness at the median line or just to the right thereof. Usually the pain is more intense and more localized than in hyperchlorhydria. and yet pain may be absent. The presence of occult blood in the stools is indicative of duodenal ulcer. The string test is here the best means of differentiation. Cholecystitis, cholelithiasis and appendicitis be included among the more important conditions which are likely to be confused with hyperchlorhydria

Prognosis.—The prognosis in hyperchlorhydria is good provided the underlying etiologic conditions are recognized. The over-acidity may continue indefinitely and becoming aggravated from time to time, it may alternate with lowered acidity and eventually constant subacidity or anacidity may prevail even without any striking change in symptoms. Usually, however, there is an abatement of the symptoms when the acidity decreases.

Treatment.—The therapeutic indications fall into two divisions: (1) measures directed toward the relief of gastric symptoms, and (2) measures suited to the gen-

eral repair of the nervous system and the removal of reflex or local causes of irritation. The measures of the second class are, with slight modifications, applicable to the other forms of functional disturbances of the stomach. Symptoms may be relieved in some cases simply by correction of diet. The aim is to give the proper food at such time that the excessive gastric secretion may combine with it, thus lessening the proportion of free HCl. This results in diminished irritation of the gastric mucosa and a mitigation of the pain and motor symptoms. Naturally, the food selected should be nonstimulating in character, should be carefully prepared and relatively free from organic acids. Condiments. carbonated waters, tea, coffee, and alcohol are best omit-Three principal meals should be taken, at eight, one and six o'clock respectively, and once or twice between each meal there should be taken fluid or semi-fluid food such as hot milk, porridge, gruels, or if it agrees, a raw egg. Meat juices and broths are undesirable, as they stimulate a greater secretion of gastric juice. Breakfast may consist of porridge, two underdone eggs, stale bread, toast, or zwiebach with an abundance of butter. For the mid-day meal may be selected purée of potatoes or peas, boiled rice with cream or butter, and perhaps sugar, fresh fish, preferably boiled, and custard or milk pudding of any sort. The evening dinner should consist of tender steak or chops, boiled meat or fish; soup without stock, potatoes boiled, baked or mashed, boiled squash, boiled lettuce, or other young, tender, very fresh. cooked salad. Uncooked oils, butter or cream may be used freely. In some instances it is well to give pure olive oil between meals in teaspoonful doses. Ices are to be avoided, water should be taken abundantly and not too cold. Before going to bed the patient should take

hot milk, or a thin gruel, or a raw egg. It is necessary to keep the intestine active and to secure full secretion from the liver. In many cases it is desirable to have the patient take on waking in the morning from two to four teaspoonfuls of Rochelle salts or sodium sulphate. With some patients better results are obtained by taking one-half tumblerful of Friedrichshall water, a bottle of Hathorn, or a wine-glassful of Abilena, or Hunyádi water. Experience teaches that a mixture of salines is a matter of some importance. We do not know why one combination suits one person and not another, but such is the fact. When the symptoms are not entirely relieved by this method of diet and the administration of salines, it is necessary to prescribe some mixture that acts as a gastric sedative and antacid. A typical mixture of this kind is the following, which I have prescribed for many years:

#### GASTRIC SEDATIVE No. 1.

7	_
ı	•
a	

Cerii oxalatis	1.00—(gr. xv)
Bismuthi subcarbonatis	
Magnesii carbonatis	4.00—(3 i)
	3.6

Sig.—A teaspoonful stirred into one-half glass of water.

This should be taken between meals, or at intervals between the taking of milk or gruel and the meals; in other words, at such time as the acid tide is at its height and when the symptoms are most in evidence. It is better to anticipate the gastric distress, to act with the first suggestion of its recurrence than to delay until the distress is acute. Some patients require a remedy more distinctly alkaline, in which cases an occasional dose of sodium bicarbonate, 1 to 4 gm., well diluted, should be given. This plan of treatment almost invariably gives relief, especially where the bowels are constipated.

Sometimes the action of the magnesia is too laxative, and for this reason it is necessary to resort to some form of gastric sedative that will quiet the bowels; as for instance:

## GASTRIC SEDATIVE No. 2.

Ð	CASIMO DEDATIVE NO. 2.			
*	Cerii oxalatis			
	Cretae preparatae 4.00—(5 i)			
	Carbo lignis	L.		

Sig.—Teaspoonful well stirred into one-half glass of water.

This may be used in lieu of the preceding prescription, or it may be alternated therewith according to the activity of the bowels. These prescriptions may be modified to suit special conditions and individual peculiarities, but they are usually sufficient. When the secretion is excessive and the distress considerable, it is sometimes necessary to give belladonna (a vagus depressant), temporarily in doses of 5 to 10 drops of the tincture before meals. Occasionally when there is pain it is a good plan to use in connection with this small doses of aconite. from 2 to 5 drops of the tincture. In those cases which show marked nervous excitement, with considerable gastric unrest, and especially if there is mental irritability, it is wise to conjoin with the gastric sedative, or other remedies that seem indicated, bromids in moderate doses. These are best given when the stomach is relatively empty. The potassium, sodium or strontium salts may be used, especially the strontium, of which 10 or 15 grains should be taken well diluted.

In cases that prove rebellious to other treatment, immediate relief sometimes follows the employment of nascent hydrogen peroxid. This may be used either in the form of "Euzone" (sodium perborate), or the peroxid of magnesium. Of these from half a gram to a

gram (gr. 7½ to 15) may be given in 4 ounces of water about an hour before meals. Lavage is not usually necessary in hyperchlorhydria, yet exceptionally there will be found a case that is greatly benefited by it. Prolonged irrigation of the stomach with a weak solution of boric acid, followed by the introduction of 3 or 4 ounces of water in which there has been stirred 2 to 4 grams of bismuth subcarbonate often proves very grateful to the patient. Sometimes a final irrigation of the stomach with a one per cent solution of resorcin is soothing to the stomach, and in comparatively rare cases great benefit follows the irrigation of the stomach with a solution of nitrate of silver 1-3000 to be followed by a thorough rinsing with normal saline solution.

GENERAL TREATMENT.—It is most important to secure for the patient mental rest, calm sleep and regular exercise in the open air. These measures alone are often entirely successful without the use of drugs. Many times we find the prompt disappearance of symptoms when the patient goes to the mountains or takes a long vovage; especially is this true when he conforms to a reasonable diet. Sometimes the symptoms disappear after careful correction of eye-strain, often even when the error is slight. This is especially true when there is irregular asymmetrical astigmatism in those using the eyes unduly, especially in those who strain the eyes by looking sharply at distant objects; for instance, school teachers, locomotive engineers, trolley men, etc. Great benefit attends general hygienic measures, such as massage, medical gymnastics, cold spinal douches, needle baths, etc. These measures must be prescribed thoughtfully, with care not to fatigue the patient and not to produce over-stimulation that may be followed by depression. Finally, it may be repeated that all efforts for complete cure may prove unavailing unless the sources of local irritation that reflexly disturb the secretion, are removed.

# Hypersecretion; Gastrosuccorbhea; Reichmann's Disease

Reichmann's disease is regarded by many as the result of pyloric obstruction, transient or otherwise, which delays the onward passage of the gastric juice. That this is often true there can be no doubt. The trouble is a result of over-irritability and spasm at the pylorus. In some cases the hypersecretion is probably independent of the state of the pylorus. This hypersecretion may be continuous or intermittent, gastrosuccorrhea continua sine periodica. Of the etiology, aside from spasm of the pylorus, little is known excepting that there occurs a perturbation of innervation in which the vagus nerves that turn on secretion are in a state of abnormal excitement.

The subject is more fully discussed in the chapter on gastrosuccorrhea (page 481).

# Hypochlorhydria and Anacidity

In healthy individuals the secretion of HCl falls at times far below the average. In the young this is usually a temporary matter. Should it persist, showing little effect from a more stimulating diet, the condition is called hypochlorhydria.

Etiology.—The condition is secondary to general or local depression of the nervous system. The lack of secretion appears to be the outcome of deficient vagal nerve impulses or to an excess of sympathetic nerve impulses to the secreting glands of the stomach; or, if we adopt a more recent view, a deficiency of hormone that

excites the secretion of gastric juice. In certain cases, as before stated, there occurs an oscillation of the gastric secretion in which a period of exaggerated secretion is succeeded by its great falling off. The precise causation of these variations is not easy to state. A low gastric secretion frequently follows a prolonged acute illness in which case the condition evidently depends upon the general depression of vitality.

Diagnosis and Differential Diagnosis.—Contrary to the old conception of impaired digestion, there are fewer symptoms occasioned by low than by high secretion of gastric juice, However, the appetite is dulled, or if it remains good, and large meals are indulged in, the patient suffers from a feeling of gastric distension or weight and from gaseous eructations. Low acidity may be accompanied by gastric atony and then the condition produces symptoms that are more distressing. If improper foods are taken, fermentation may occur and gastritis may follow: yet ordinarily, the manifestations result more from atony than from lowered secretion. On the other hand in cases of long-continued low acidity, the motor function of the stomach becomes exaggerated, and then the gastric symptoms decrease, although intestinal digestion may suffer correspondingly. Cases of this sort approach in nature achylia gastrica. They are given more ample consideration under the head Achylia Gastrica (page 604). The diagnosis can only be made by often repeated examination of the stomach contents made under varied conditions; for instance, after rest, after exercise, after periods of worry, etc. A case would not be considered one of hypochlorhydria unless the total acidity was found quite uniformly at a low point, say from 5 to 25, and occasionally disappeared entirely. As a rule the secretion of ferments is in ratio with that of

the acid, although this is not always true. The examination of exceptionally healthy individuals reveals the fact that in a certain number a low acidity is the rule and this without any symptoms whatever, apparently without disturbance of health. This should be borne in mind in making a differential diagnosis and we should also attempt to draw a line between cases of temporary functional depression in secretion and those cases of continued absence of secretion, both acid and ferments, which go under the name of achylia gastrica. Finally, we should distinguish between functional failure of secretion and a failure which is secondary to gastritis. In the latter mucus is usually present in the stomach contents besides other evidences of inflammation.

**Prognosis.**—The prognosis is favorable so long as the mucosa remains in a normal condition.

Treatment.—The treatment should be that which is likely to restore the general health. Anemia should be combated, muscular and nervous power increased; hence long hours for sleep, and short hours of work should be insisted upon. The establishment of a permanent low secretion or anacidity should be earnestly avoided, and it is therefore advisable to keep these cases under treatment until the power of the stomach is restored. alternating hot and cold body douche, the cold spinal douche, massage, especially of the abdomen and of the spinal centers, deep breathing exercises, and other medical gymnastics are of real value, and a life in the open air should be urged. Temporary improvement often follows local treatment of the stomach. This may take the form of intragastric faradization, alternating hot and cold, intragastric douche, or the administration of drugs calculated to stimulate secretion. There is benefit from the simple bitters, nux vomica, small doses of alcohol before meals, the taking of animal broths on an empty stomach, and local stimulation in the form of condiments, aromatics and carbonated waters.

A dessert-spoonful of vermouth (Italian), with a little shaved ice, or a small cupful of very hot bouillon with red pepper may be taken before meals or a glassful of ginger ale with meals.

## MOTOR DISTURBANCES

In the stomach, as well as in other portions of the digestive tract, the motor disturbances surpass others in importance. There is a wide range in the symptoms from gastric tormina or spasm on the one hand to complete atony on the other. Gastric unrest shows itself by conscious movement of the stomach, usually accompanied with gaseous eructations which are favored by the patient in the hope of obtaining relief. The unrest is usually and erroneously attributed to gas. The condition in fact is the result of motor irritability, often associated with hyperesthesia and with over-tonicity.

Excessive gastric motion, usually with violent upward escape of gas, sometimes takes the form of distinct spasm of the stomach and is a disagreeable symptom often associated with excessive secretion and sensations of distress. In over-tonicity, the stomach may empty itself slowly for the reason that the pylorus remains in a state of over-contraction, a condition which may show itself in the cardia as well. It is a singular fact that one part of the stomach may persist in over-tonicity while other parts remain uninvolved. Illustrations of this are cardiospasm, spasmodic biloculation and spasm of the pylorus. It is true that these symptoms may be excited by local irritation, for example in case of ulcer or perigastric adhesions. It is not easy to explain the hypermotility

and excessive tonus in many of these cases. As is true of gastralgia, so also gastric spasm may originate from irritation in regions remote from the stomach. Although it is excited by contiguous irritation, the most common cause of pyloric spasm, for instance, being disease of the gall-bladder, yet I have recorded instances which have depended upon retroversion of the uterus, stone in the bladder and irritation in other comparatively distant parts. It is only necessary to recall the frequency of motor disturbances of the stomach in pregnancy to illustrate my meaning. Some would explain these facts by assuming the presence of a vagotonic state.

Symptoms and Signs.—Gastric hypermotility and overtonicity may be recognized by the sensation of tension, or of movement in the stomach of which the patient complains. Almost invariably relief is sought for supposed gastric fermentation, which as a matter of fact rarely obtains. There is frequent eructation of gas, less often regurgitation of sour fluid which is supposed to depend upon fermentation. The subjects of the disorder are so insistent upon the presence of fermentation that they frequently convert the physician to their view. There is usually a history of a long course of treatment by antiseptic drugs. It seems well to emphasize this statement. By the introduction of the stomach tube, while keeping the proximal end submerged in water it may be easily shown that the stomach contains no more gas than is The radiograms of the normal stomach during digestion reveal the invariable presence in the fundus of superimposed gas, like a huge bubble. Examination of the stomach contents demonstrates that there are no fermentation and no organic acids and only the usual bacteria are present. The sour fluid regurgitated is merely the gastric juice. At times there is an hysterical side

to the case in which a large volume of gas is noisily discharged; this is quite characteristic. One may make a diagnosis of these cases through hearing for a few moments these violent expulsions. Such manifestations result from the unconscious swallowing of air. (See Aerophagy.) As a rule the symptom of eructation of gas is less conspicuous than this, yet the account of the patient in describing his case is emphatic as to the importance of gas. This symptom is commonly the result of motor irritability. Where fermentation actually exists and where gas develops in pathologic quantity, there does occur the symptom of eructation, but it is remarkable how infrequently we encounter this true excess of gas in actual practice. Even in gastritis, the symptom of belching depends more upon the irritability of the stomach than upon the active evolution of gas. Occasionally, the stomach is very resentful to the stomach tube. At such times there is vigorous involuntary contraction of the abdominal muscles, but apparently the stomach is also associated in the contraction The stomach contents is ejected with unusual force and slight injury to the gastric mucosa results from the pressure upon the distal end of the tube so that abrasions of the part as shown by slight bleeding, may occur. This may be obviated by reassuring the patient and demanding his coöperation and self-control. After a few examinations with skill in the technique, there will be little accompanying distress. A large tube, having a rounded extremity. should be used, thus shortening the examination and avoiding the source of trauma. So the examiner may acquire needed information, with little disturbance of the patient. The diagnostic use of the stomach tube, instead of being injurious, generally has a mitigating effect upon the motor irritability.

Prognosis.—The functional motor disturbances of the stomach are as a rule easily controlled. The exceptions exist in cases of cardiospasm and more rarely in cases of pylorospasm. Atony, however, is more intractable.

Treatment.—A good part of the motor excitability is under the voluntary control of the patient. This fact is not recognized until the patient is educated not to belch and has gained an understanding of the real meaning of the symptoms. In these neurotic cases the stimulation to the stomach which attends digestion, exciting as it naturally does the physiologic movements of the stomach, sets up a strong tendency to the unnatural movements. For this reason it is sometimes necessary to soothe the stomach by the administration of a sedative mixture before meals. For this nothing is better than cerium oxalate and bismuth subnitrate. It is rarely necessary to use the more powerful sedatives like chloral. In hysterical cases asafetida in the form of the mistura, an ounce or two per rectum, is of great use. It may be given per os, but the patient will complain of the disagreeable eructations. This very fact has occasionally a beneficial side; more restraint will be used to prevent The oil of amber, valerianic acid, the eructations. validol, the essential oil of camphor or other anti-spasmodics may be required. A systematic course of hydrotherapy, medical gymnastics and occasionally intragastric applications of electricity are much more serviceable than drugs. The alternating hot and cold spinal douche and hot abdominal fomentations, followed by cold spray or affusion will assist in improving innervation and have an excellent moral effect upon the patient. The same is to be said of systematic lavage, for which very warm water may be used, the irrigation to be continued for ten minutes; or occasionally an alternation between water at

#### 174 FUNCTIONAL DERANGEMENTS OF DIGESTION

a temperature of 105° and that of 60° F may be employed. It may be necessary to eliminate from the diet certain forms of food that are peculiarly and sometimes unaccountably disturbing. Some people apparently in good health will suffer from gaseous eructations whenever certain foods are taken, and it is to be assumed that this effect would be heightened in those having an irritable stomach. It may be an economy of time to begin with very bland food, steadily increasing the variety until the ordinary diet is reached. Dr. Leared long ago called attention to the good effect of charcoal in these cases. is best to give the very finely powdered, vegetable charcoal from which moisture has been driven off by re-ignition in an iron capsule. While still warm the charcoal may be bottled and when a dose is necessary half a teaspoonful is inclosed in a wafer paper and swallowed, preferably when the stomach is empty. I am convinced of the real benefit following this method of administration. Carminatives are often of great assistance. these, ginger, peppermint, cardamom and gaultheria stand first. The following mixture is sometimes useful:

Sig.—To be taken in a little water at one dose.	
Degr.—10 be taken in a little water at the about	
Or, the following mixture:	
Magnesii carbonatis 2.00—(3 ss)	
Tincturae cardomomi compositae 8.00—(3 ii)	
Tincturae rhei	
Spiriti chloroformi 1.20—(M xx)	
Syrupi zinziberis	
Aquae carui	
М.	
Sig.—Take one or two teaspoonfuls repeatedly as needed.	

Ų,

A time-honored and useful combination consists of equal parts of the compound spirits of ether, compound spirits of lavender and aromatic spirits of ammonia; of which half a teaspoonful in a wine-glass of water may be taken as often as necessary. It will be seen that there is some inconsistency in the response to medicaments on the part of the stomach; it is a fact that while soothing measures succeed, stimulating measures, even in the same case, may also serve a useful purpose. These details of treatment may seem superfluous, but it is hoped that their consideration here may help to make clear the management of this often misunderstood condition.

#### RUMINATION

The regurgitation and remastication of food, although a very exceptional condition, is nevertheless in some cases something more than a habit neurosis. When it develops after childhood and is continued as an hysterical manifestation, it may be but a transient symptom and one that may be easily controlled. At other times it is the expression of an inherited peculiarity, perhaps atavistic in nature. Brockbank 2 reports having found the condition in four generations of the same family, and several other well authenticated instances apparently showing heredity, are reported. Some persons have no control over the act of regurgitation, while in others it may be temporarily repressed though this repression produces marked discomfort. Undoubtedly imitation exerts an influence in originating these family cases, although it has in some instances been excluded. are many isolated cases that have not been traced either to imitation or heredity. In some instances certain articles of food, especially meat, are regurgitated, yet other

<sup>&</sup>lt;sup>2</sup> Brit. Med. Jour., Feb. 23, '07.

foods, even when taken at the same meal, are not brought up. At times the taking of certain foods re-excites the regurgitation when previously it has been under control. The practice does not appear to disturb the general health, except that it is likely to induce over-self-consciousness. Some cases may be relieved by education and suggestion while others are uncontrollable. Drugs appear to have little influence upon the affection, and when efficacious it is probably through their psychic effect.

#### SPASM OF THE PYLOBUS .

In some cases it is not easy to measure to what extent spasm of the pylorus is a purely neurotic performance, and to what extent it depends upon some local irritation at or near the pyloric extremity of the stomach. By recalling the physiology of the normal closing of the pylorus, we see that normal contraction of the part occurs as the result of a reflex. When the gastric juice becomes sufficiently acid and when it carries with it those portions of the aliment already sufficiently acted on by the gastric juice, then the acid chyme is projected in short spurts through the pylorus into the duodenum. Through the action of the duodenal reflex, this acidifying of the contents of that organ stimulates the pylorus to contraction and to closure of its gates. The pyloric orifice remains closed until the chyme which has entered the duodenum becomes neutralized. When this is accomplished the action of the duodenal reflex subsides, the chyme within the stomach signals the pylorus to relax, to open the door, in fact to permit the egress of another spurt of gastric contents, and so on with orderly and sympathetic coöperation until the contents of the stomach is delivered to the intestines. When there is an obstruction in the intestines, or when there is a lesion that is very irritating, the intestine is unwilling to carry out its part of the contract. It rebels when the stomach forces work upon it and sends such a reflex of inhibition to the pylorus that this part of the stomach ceases to relax and to discharge its usual portion of the contents into the duodenum. In short, there takes place what we are accustomed to name pyloric spasm. As a result of this the intestine is enabled to rest and perhaps to recuperate; but as another result there is occasioned a stasis of gastric contents. That is, there is ischochymia. "motor insufficiency" or food stagnation. The stomach is thus embarrassed and, through various unmistakable messages, it soon makes the individual conscious of this break in harmony. There are anorexia, eructations, regurgitations, nausea and perhaps vomiting; there is dyspepsia with distress and perhaps pain.

As there are degrees of pyloric spasm, so there follow degrees of gastric stasis. Spasm may be just sufficient to delay but not to stop the stream; it may be so intense that the stream is not only stopped but when gastric retrostalsis occurs, the stream may be reversed.

The irritation that produces the spasmodic closure of the pylorus may reside in the pylorus itself when it is the seat of inflammation or ulceration; but pylorospasm in degree quite as marked may be induced without disease of that part, as has been stated. Therefore, in case of duodenal ulcer or duodenitis, of appendicitis or cecal stasis, enteritis or even colitis, we may find that the door of the stomach is closed to the escape of its acid contents into an unwilling intestine. There is an analogy between this condition and the congenital pyloric spasm and stenosis elsewhere described. Of course, pyloric spasm is often associated with hyperchlorhydria, and as the gas-

tric acidity is raised when delay in emptying the stomach occurs, it seems that there is reason for confusion as to the precise mechanism at work in these cases of functional pyloric spasm. Apparently this is an instance of an exaggerated vagotonic state. It is of little practical importance whether or not the pyloric spasm is associated with over-acidity in these functional cases. fact that spasm may be solely dependent upon neurotic causes is really the important question. It is an interesting fact that in the same patient we may discover spasm both of the cardia and of the pylorus. It would seem most natural that parts having such a high degree of functional motor activity should be liable to derangement and, as might be expected, we see analogous disturbances of motion in the urinary bladder, in the rectum and in other organs. The question is more fully discussed in the chapter on Motor Insufficiency.

In cases of pyloric spasm accompanied by over-secretion, it is possible that, in addition to the action of the duodenal reflex, the gastric hormone, or internal secretion, described by Edkins, may be responsible for a degree of the over-contractility.

An example of what appeared to be true gastric spasm occurred in association with extreme anemia following the vomiting of pregnancy.

Case No. V. Vomiting throughout Pregnancy, Severe Post-Partum Hemorrhage, Grave Anemia, Transfusion, Great Improvement, Pyloric Spasm, Ultimate Recovery. -Mrs. Y., aged 30, had vomited from the beginning of her pregnancy, which event was attended with great loss of blood. Following delivery the vomiting continued without apparent reason. She entered the Buffalo General Hospital and was found to have a red count of only 800,-000, white count 1500, hemoglobin 18 per cent. Drs. Busch

and Eschelman transfused by artery and vein anastomosis; the "donor" was the patient's sister, and her blood was shown to be non-hemolytic. The exsanguinated surface of the patient pinkened; that of the "donor" became pale, the pupils dilated and faintness was experienced, when the operation was discontinued. The patient's hemoglobin rose to 30 per cent and the blood count was The patient rapidly gained and in a few days the red count was 2,000,000 and the hemoglobin 39 per cent. Improvement was checked by a recurrence of the vomiting. The radiogram revealed complete obstruction (Radiograms V, VI, VII.) Apparently at the pylorus. nothing was discharged through the pylorus for several days. Because of the curious markings of the radiogram, it was at first feared that we were dealing with a carcinomatous infiltration of the lower end of the stomach. sequent radiograms dispelled this fear. Through the hypodermic administration of atropin with the view of cutting out vagal excitement, the pylorus relaxed and the patient took food immediately in abundance and all the gastric symptoms disappeared quite magically. This was a case suitable for the use of adrenalin but at the time its action in overcoming gastric spasm was not understood. The accompanying reproduction of the radiograms taken of this case should be of special interest for they not only show the possibility of mistaking a purely functional condition for a gross anatomical lesion, but they also show the striking changes that take place in the contour of the stomach under the condition of pylorospasm. tient is now in perfect health.

### CARDIOSPASM AND PYLOROSPASM

It is not unusual to find individuals complaining of slight difficulty in swallowing certain foods which seem 180

to lodge at the end of the esophagus. This slight delay is overcome by a swallow of water and is only of moder ate inconvenience at certain times. These cases are not sufficiently severe to be classed as simple or true cardiospasm, nevertheless they seem to belong in that class. I refer now to individuals who do not consider themselves ill, but who suffer more frequently from this symptom than is the case with ordinary people. It is a common experience to find that the stomach tube is grasped by the contracting cardia so that it is well to wait a little before attempting to introduce the tube into the stomach. After a moment this tension subsides. When the cardia is over-irritable, it may relax suddenly while the stomach contracts spasmodically, whereupon a small amount of gas within the stomach may be discharged with considerable violence. At other times the stomach contracts in a spasmodic manner, but the cardia refuses to open and there is thus produced a somewhat painful sensation of tension or pressure. When an unsuitable meal has been taken, when fermentation occurs in the stomach, and considerable gastric flatulence results, it may be impossible for the patient to dislodge the gas. the cardia refusing to relax. In rare instances gas is evolved in the stomach in large quantities and does not escape from either cardia or pylorus. The upward pressure round the heart causes marked disturbance of circulation and even syncope. The factors at work in producing this condition are probably related to the causes of acute dilatation of the stomach. Sudden death is believed to have resulted in some cases from the overdistension of the stomach with gas and from the resulting abdominal shock which this induces. Careful autopsy studies apparently warrant this conclusion. From the observation collected it seems justifiable to attribute con-



Radiogram No. V.—CASE No. V. GASTRIC SPASM. Thirty minutes after bismuth meal. Note the irregular and blurred outline of shadow at prepyloric area, suggesting infiltration of the stomach walls,



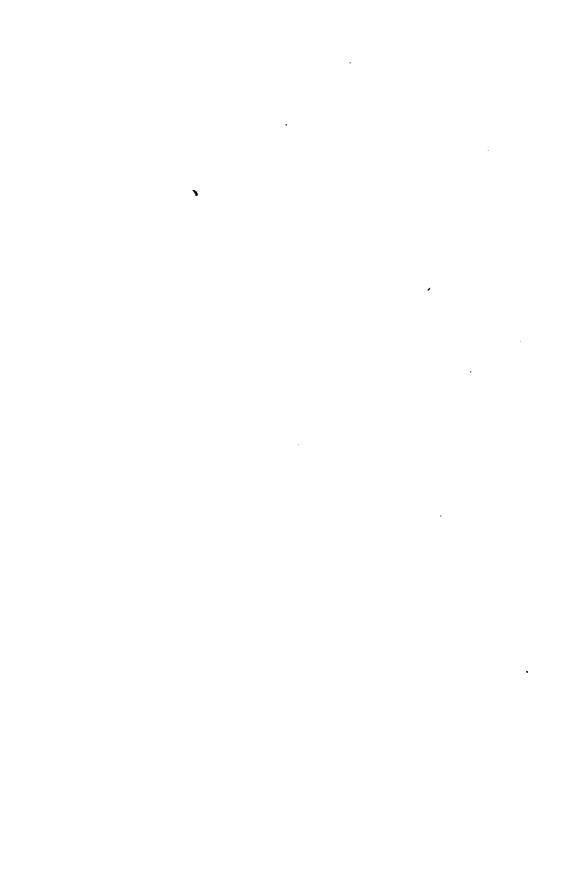
Radiogram No. VI.—CASE No. V. GASTRIC SPASM. One and one-half hours after bismuth meal. A blurred aspect of the shadow noted in Radiogram No. 7 appears in this. No bismuth has passed the pylorus.



Radiogram No. VII.—CASE No. V. GASTRIC SPASM. Taken on another day, four hours after a bismuth meal. No bismuth has passed the pylorus. Pyloric obstruction complete from spasm.



Radiogram No. VIII.—UNUSUAL CASE OF HOUR-GLASS OR BILOCULAR STOMACH. Case came to operation.



siderable importance to the temporary spasm of the cardia as well as of the pylorus.

Symptoms and Signs.—The symptoms of temporary spasm of the cardia have already been stated. They consist in the inability to discharge gas from the stomach through the esophagus and the temporary obstruction to the swallowing even of liquid. There is also occasional distension of the stomach with gas and difficulty is experienced in passing a bougie into the stomach.

The symptoms of pylorospasm correspond somewhat to those of hyperchlorhydria. The contracture is accompanied by gastric distress, and the patient becomes conscious that the stomach is not normally emptying itself. Often there is regurgitation of gastric contents having the taste of food taken hours before, and occurring when the stomach should be empty. Occasionally pain is excited as the result of the spasmodic contraction of the pylorus, and the accompanying hyperacidity, even when we have reason to believe that no ulcer or gastritis exists. The stomach tube introduced seven hours after a meal will show ischochymia; that is, food will be found at a time when the stomach should be empty. The persistency of the spasm may be somewhat roughly measured by the delay which occurs in the emptying of the stomach. A more precise method is the lactose test meal of Roberts.3 Usually there is found increase of acid gastric juice, and the symptom complex of Reichmann's disease. Radiography may assist in the diagnosis but the warning is given not to be convinced prematurely. Repeated exposures at strategic moments are useful but misconception is otherwise almost inevitable.

<sup>&</sup>lt;sup>8</sup> Am. Jour. Med. Sci., Nov. 1912.

**Prognosis.**—The prognosis in these cases is usually good, although certain persons have a tendency towards the recurrence of the symptoms, particularly when undergoing great nervous fatigue, accompanied by indiscreet eating. Cardiospasm, long neglected may involve serious denutrition; as a complication, pyloric spasm may become a dangerous factor.

Treatment.—A plain, unstimulating diet and frequent and prolonged lavage are the best means of procuring immediate relief. The overacidity of the gastric juice should be neutralized and the spastic condition may be overcome by sodium bromid and other antispasmodic Chloretone or chloral are very useful in remedies. some cases. The milk of magnesium and of cerium, either alone or blended, taken frequently between meals. tend to prevent the recurrence of the symptoms. Meantime attention should be given to the general health, for it seems probable that the spasm depends upon an over-excitability of the autonomic nervous system and certain spinal centers, as well as upon derangement of the sympathetics. This plan of treatment applies to pyloric spasm as well as to the milder forms of spasm of the cardia.

The relief afforded gastric distress by the taking of food has been ascribed to the resulting modification of the gastric chemistry and hence decreased irritation. It has been observed that this relief is often instantaneous, as well as temporary. A stretch of imagination is required to believe that gastric acidity is thus suddenly reduced, which must be the case if by that means, lessened acidity, the relief is produced. A more rational view is to suppose that the distress which attends hyperchlorhydria and allied conditions, is in fact the result of exaggerated gastric tonus, and that any influence that

operates suddenly to overcome this tonus gives immediate relief to distress and pain.

Now it has been shown by physiologists 4 that as soon as the food reaches the cardia, the receptive stomach loses instantaneously its tonus, the intragastric pressure falls to zero and the food proceeds into the relaxed stomach. This mechanism which quickly though temporarily overcomes the gastric tonicity, also overcomes the stretching of irritated tissue, and probably is the factor which is accountable for the relief to gastric distress and to "hunger pain" which follows at once upon swallowing food.

From experimental work it is found that adrenalin, administered subcutaneously, leads at once to cessation of tonus, to the relaxation of the stomach and the intestine.

A limited personal experience indicates that adrenalin will prove to be of clinical value in relieving gastric spasm.

# DIMINISHED GASTRIC MOTION, HYPOMOTILITY, GASTRIC ATONY

Temporary decline in the motor power of the stomach is common. It occurs from time to time in all individuals, but it may appear in a severe form and continue indefinitely, constituting in itself a real ailment. Gastric lavage from time to time through an extended period shows that with little variation, the stomach is emptied tardily. When it reaches this state, the condition is spoken of as gastric atony. This condition may come about from nervous depression, such as follows severe mental shock, bodily injury or surgical operation. It is not an unusual sequence of acute illnesses, especially the

<sup>&</sup>lt;sup>4</sup> Cannon: "The Mechanical Factors of Digestion," p. 237.

infections, in which it is highly probable that we are at times dealing with degeneration of the walls of the stomach, in addition to the depressing effects of infection in other portions of the body acting upon the stomach through lowered innervation, failure in blood pressure, etc.

In the cases of advanced and prolonged atony of the stomach, dilatation of the organ takes place, the gastric walls become thin, their muscle attenuated and the blood supply impeded. Such a condition is sure to interfere seriously with digestion and with the general health. There are many cases of temporary depression of motion, which must depend upon transient causes, as the symptoms are readily controlled and the motion of the stomach is found to be restored. Between these two types of cases, there are found to exist all gradations.

The subject of gastric atony receives consideration elsewhere (page 488) but it has been mentioned here to indicate the relationship existing between true atony and a temporary or transient state dependent upon nervous causes.

Treatment.—Though it seems inconsistent, certain remedies that are employed in gastric over-motility are successful in overcoming depression in motion. In other words, stimulants, carminatives, heat and cold, electricity and massage are important in giving strength to the motor function of the stomach. In lowered as well as in excited motility the fault lies in a loss of physiologic harmony. To restore this harmony the same measures may succeed, regardless of whether there is little motion or spasmodic action. Of course this statement does not apply to the use of sedatives given to quiet local irritability in case of motor excitement.

#### NEUROSES AFFECTING SENSATION

#### GASTRIC HYPERESTHESIA

One of the most common of sensory disturbances of the stomach is that known as hyperesthesia. It is a local manifestation of general irritability, and is comparable to general or to other local instances of hyperesthesia, manifestations of hysteria, or functional weakness. It may or may not be associated with psychic phenomena. It is sometimes accompanied by other types of nervous derangement. This is quite commonly the case and careful examination as a rule discovers evidence of nervous depression or neurasthenia. The importance of recognizing gastric hyperesthesia was shown by the late Dr. Steel of Philadelphia.

Etiology.—The affection is prone to occur in those having a lowered nervous resistance and is excited by sleeplessness, anxiety and fretting, by eye-strain, indoor life and depressing influences in general.

Symptoms.—After eating, the patient soon feels gastric distress and sensations of burning, weight, distension, and sometimes soreness of the stomach, as if the mucous membrane were over-delicate. The symptoms are usually in proportion to the stimulating character of the food. Bland liquid, or semi-solid foods may be tolerated, while solid foods, especially if they are not well masticated excite the symptoms. The victims of this trouble often become notional. Having morbid fear of eating they restrict themselves more and more until the hyperesthetic state is aggravated as a result of lowered nutrition from starvation. At other times seemingly they unreasonably complain of particular articles of diet that would appear to be inoffensive. Some are especially averse to hot foods, others to cold: some are averse to 186

salt, others to sugar and still others to acid. The imagination undoubtedly plays an important rôle in the matter, and it is not easy to distinguish the real from the fancied disturbances. It is a mistake to regard these patients as merely the subjects of their own fears. Important as this side of the question may be, there is no doubt but that much of the complaint is based upon actual sensations. Almost always there is an accompanying motor irritability of the stomach, and patients state that the stomach contracts painfully after eating. There are frequent eructations of small quantities of gas and possibly regurgitations of stomach contents, often lacking in acidity. The gastric secretion is not of high acidity. and when the HCl is found above the standard it is impossible to differentiate these cases from those of hyperchlorhydria. Doubtless in some instances the two conditions are associated, in which case the symptoms of hyperchlorhydria are more marked than would be expected from the acidity present. There is usually moderate, diffuse tenderness over the epigastrium, and the patient often complains of soreness in the stomach upon the introduction of the tube.

The study of the stomach contents in uncomplicated hyperesthesia shows no evidence of gastritis; however, in some cases of gastritis hyperesthesia may be present. The hyperesthetic state may have engrafted upon it not only other types of nerve disturbance, but also various diseases of structure as well. Gastric hyperesthesia is not a rare affection, the element of over-sensitiveness entering into a variety of other morbid conditions.

**Prognosis.**—The course of the affection will depend upon the underlying neurasthenic state. Many patients suffer from the condition for years under the misapprehension that there exists a weakened digestion. The

symptoms can be relieved, however, by general measures hygienic and otherwise, by means of which a dilapidated nervous system may be restored to good behavior.

Treatment.—Even at the risk of repeating too often what has already been insisted upon, that functional stomach trouble requires general treatment, it must be pointed out again that much of the lack of success in relieving dyspepsia lies in the absence of a full realization of this fact. One of the first steps therefore is to convince the patient that he has not a stomach disease. that, in point of fact, his stomach is able to perform the necessary functions even though some distress is excited thereby. This reassurance is of great help to the patient; and his attention being directed towards his general health, he experiences less gastric disturbance. Next the patient must be taught that starvation is bad for him and time should be taken to convince him that the constantly increasing restrictions in diet which have been practiced constitute an important part of the trouble. Thus while building up the general health and nervous resistance we should deliberately reëducate the patient's appetite and see to it that a full number of calories are daily ingested. This should be done gradually, but without delay, because the patient's confidence should not be shaken. The stomach should be given rest, the meals separated by a period of about five hours; yet this rule may not be applied immediately to those who have long practiced the habit of "sipping" a little every hour or two. Meantime assistance is gained by both general and local measures calculated to soothe nerve irritability. Thus there should be given a daily full warm bath and there should be applied those massage movements that are successful in quieting irritable nerves. To prevent too great relaxation following this 188

treatment, the graduated cold spinal douche, general faradization or the descending continuous current over the spine may be used. A sojourn at the seaside, or some quiet place removed from the distractions of busy life, is to be considered. A measure which acts both generally and locally and which is almost indispensable in the beginning of the treatment of some cases is the administration of the bromid salts, especially strontium, shortly before eating. I usually prescribe strontium bromid, 1.00 gm. (gr. xv) and subcarbonate of bismuth, 1.00 gm. (gr. xv) to be given in wine-glass of water. Cherry laurel water and chloroform water in equal parts, one teaspoonful, may be taken from time to time as the symptoms demand. Occasionally carminatives like mint, ginger, cardamom or anise, given in watery solution with chalk mixture or combined with bismuth and cerium oxalate, may be given to subdue symptoms. The ingenuity of the practitioner will suggest other measures suited to the peculiarities of the patient. Meantime the restoration of the nervous system by drugs must not be omitted. After meals, there should be given the glycerophosphates of lime and soda each 0.18 gm. (gr. iii). The calcium salt is especially valuable in females of a lymphatic type who suffer from morning languor, especially near the menstrual periods. It should be remembered that in some persons glycerophosphates may increase nerve irritability. As these patients are often anemic, it is advisable to prescribe some form of iron, at times in combination with arsenic. The tincture of the chlorid of iron or the freshly made Blaud's pills with arsenic, or natural iron and arsenic waters, like Rencegno or Levico, often serve a good purpose. Arsenic at times causes gastric discomfort in which case it must be given in small doses well diluted, or arsenic and iron may be administered subcutaneously. Weekly estimation of the hemoglobin should be the guide for the continuance of the chalybeate. If a laxative becomes necessary an occasional saline purgative may be prescribed, but as a rule the best results follow the systemic irrigation of the colon. As improvement in general health occurs the attempt should be made to regulate the bowels by diet by giving from 4.00 to 10.00 gm. (3i-3ii ss) of finely divided agar-agar and by securing a regular habit in evacuation. Medical gymnastics including abdominal massage contribute to this result.

#### Paresthesia Gastrica

Closely allied to hyperesthesia is paresthesia, or the presence of unnatural sensations of various kinds. The condition is seen in the psychasthenic and morbid imagination greatly increases the local discomfort. The description of the morbid sensations varies with the individual. A complaint of the sensation of coldness in the epigastrium is common, as is also that of trembling. Occasionally there is the time-worn belief of the presence of some living creature in the stomach, an idea which is not limited to the ignorant and superstitious.

A constant craving for food and a sense of emptiness in the stomach is frequent; in an exaggerated form these symptoms constitute the condition known as bulimia. The patient not only has an unnatural craving for food, but the psychic accompaniment is noticeable, so that the patient eats in abnormal manner. The quantity of food taken is sometimes enormous. Occasionally this is rejected by vomiting, but this does not abate the hunger of the subject; he vomits and then asks for more food. I have seen this in small children. Frequently the stomach undergoes dilatation, but probably without marked

190

thinning of the muscle coat. I have not encountered the condition joined to a case of real gastrectasis with food stagnation.

Symptoms and Signs.—The subjective symptoms constitute all that we know of this condition. The diagnosis must be made by excluding true insanity and the craving which occurs in case of over-motor activity of the stomach with too rapid emptying. Practically, the diagnosis has to be made by the history and by exclusion.

**Prognosis.**—In the simpler expression of paresthesia, the prognosis is good; yet in cases in which the psychic side is pronounced, where in fact we have to deal with a psychosis, the condition is more rebellious to treatment.

Treatment.—In the severe cases with true psychosis. we must resort to methods of education, discipline and encouragement. I have no doubt that much can be accomplished in some apparently intractable cases by means of psychotherapy. It is difficult to control the patient for a sufficient length of time. The simpler cases, those having merely the presence of unnatural craving, the sensation of trembling or coldness, or the feeling that there is present a foreign body, are usually relieved by strong suggestion, by winning the patient's confidence and proving by means of the stomach tube that the sensations are groundless. For instance, when the patient insists that his stomach is empty it may be shown that it has an ample content. When there is fear of the presence of a living or other foreign body, the idea may be dispelled by the use of the esophagoscope. It is not wise to argue with these patients; it is better to demonstrate to their satisfaction that what you say is true, whereupon the moral treatment is much more effectual. Of course in the meantime everything possible should be done to raise the general health to its highest plane.

#### Anorexia Nervosa

Loss of the appetite arises from so many different sources that the existence of mere nervous anorexia is easily overlooked. It is a very frequent derangement and it is more especially seen in women and in young girls.

It may be said that the condition is but an expression of a general functional depression and that it is unnecessary to treat of it as a special affection. much truth in this assertion, yet nervous anorexia is a very important manifestation of general depression and unless it is intelligently dealt with it offers a serious obstruction to restoration of the patient's health. Nervous loss of appetite often supervenes upon grief, disappointment or psychic shock. It sometimes follows trauma or acute illness. Whenever an individual becomes over-selfconscious, especially when to this is added injury to amour propre, it is quite the usual thing for appetite to disappear. Anorexia may come on acutely after a mental shock. Through its persistence it favors other nervous affections and may leave the patient unresisting to infective and constitutional diseases.

Symptoms and Signs.—There occurs a complete loss of appetite, often a great disinclination even to taking liquids. There is complaint that food cannot be swallowed, perhaps the suspicion of an obstruction in the esophagus. Usually there is discomfort following the taking of food. The more eating is urged the more the patient rebels. As a result there is chlorosis, glandular secretions fall off and the excretions of the body become unnatural. There is acidosis, the tongue becomes coated, the breath offensive and a disagreeable taste is described. General lassitude develops, there is marked loss in weight and

in muscle power. With this is associated mental depression and introspection.

In the foreground of the picture other functional disturbances appear, such as insomnia, bad dreams, headache or dizziness, pruritus local or general, occipital headache, lumbar backache and catamenial derangement. Constipation usually aggravates the situation. Partly from insufficient drinking of water the urine becomes concentrated, uratic or phosphatic, a condition which is an index of the unphysiological state of the general nutrition. This latter shows itself in arthralgias, neuralgias and tenderness upon pressure, and the diagnosis of neuritis is often made mistakenly. I have seen cases which have continued for years, having undergone endless treatment for various supposed diseases when really there was required only the establishment of proper habits of living in order to procure relief.

Prognosis.—Usually the condition is readily removed by proper management.

Treatment.—In the beginning it is all-important to exclude other diseases. We must be certain that we are not dealing with tuberculosis, mental disease or some other hidden pathology. When these have been excluded. we should institute a disciplinary treatment, and in this the personality of the doctor is of more importance than his drugs. Before taking up the question of eating, it is best to clear the way by reëstablishing the secretions. The skin must be made active, a full purgative given, and the colon subsequently treated by means of high and stimulating or antiseptic irrigations. For this I find that an infusion of inspissated oxgall, 4.00 gm. (3i); warm water, one liter (Oii), or a weak infusion of chamomile or mint are very satisfactory.

Massage and systematic active exercise should be prac-

ticed as soon as possible, and the patient should be made to use the hands in some work not requiring too close use of the eyes; basket-making and wood-carving are illustrations. In the beginning, the prescribed amount of semi-solid food must be taken at definite hours and a given amount of liquid should be drunk each day. obstinate cases it is best to begin by lavage, followed by the introduction through the tube of a pint or more of nutritious semi-solid food. When the patient becomes convinced that food can be tolerated, it is comparatively easy to increase the amount and to bring about regularity in meals. By positive instructions without too much talking, by firmness, and by showing results in improvement in weight, it will soon be possible to raise the general nutrition to a point where there is greater safety. With patience and time the patient will soon show an appetite for certain meals and very soon three regular repasts will be enjoyed. At times the food gives rise to much discomfort in which case material assistance follows the administration before meals of a soothing mixture like the following:

```
      R
      Tincturae aconiti
      0.12 (gtt. ii)

      Strontii bromidi
      0.60 (gr. x)

      Aquae gaultheriae
      ...

      Misturae acaciae
      ...

      M.
      Sig.—Take in a wine-glass of water.
```

To soothe the irritable nervous system, the following prescription given three times a day is of service:

Apparently some patients are benefited by the simple bitters before meals in which case the infusion of chiretta 30 c.c. (3i), infusion condurango, 30 c.c. (3i), or infusion of quassia 15 c.c. (3ss), or the Italian vermouth, a table-spoonful before meals are useful. Gastric activity sometimes needs stimulating, in which case I sometimes give five-drop doses of dilute hydrochloric acid before meals or a pill containing:

```
\( \begin{align*} \text{Quininae hydrochloratis} & ... & 0.06—(gr. i) \\ \text{Oleoresinae piperis} & ... & .01—(gr. \frac{1}{2}) \\ \text{Tragacanthae} & ... & 0.12—(gr. ii) \\ \text{M.} \end{align*} \]

\( \begin{align*} \text{M.} & Sig. — Dose one pill before meals. \end{align*} \]
```

It is sometimes better to avoid drugs entirely and especially hypnotics. The so-called "drip sheet rub" at night followed by a glassful of the infusion of linden flowers (thé de tilleul), or a half teaspoonful of the compound spirits of lavender in a glassful of hot water, will generally induce sleep, but if not other simple measures must be selected. It is important to secure a good capillary circulation, and especially to overcome the coldness of extremities and cerebral congestion that the patient so often complains of. The cold spinal douche followed by massage, spinal pressure with vibration, is most important and I am in the habit of prescribing a special, very brief, ice water immersion of the feet one after the other, accompanied by brisk scrubbing and followed by thorough drying and quick dressing of the feet in warm stockings. When strength permits, the patient should engage for a short time regularly in some active game. providing exercise and recreation, for instance, tether ball, Indian club, or fencing. When practicable, nothing is better than daily hill-climbing, progressively increased. the patient dressed suitably for the weather. The attempt should be made to produce a gentle perspiration by physical activity. In feeble cases, it may be necessary to use a cabinet bath, perhaps followed by a brief full needle bath two or three times a week. It will be seen that the plan of treatment here described is in reality as well suited to other forms of functional disturbance as to nervous anorexia. It is for this reason that the treatment proves so successful.

## BULIMIA, HYPEROREXIA—POLYPHAGIA, AKORIA

The terms bulimia and hyperorexia have a different significance from polyphagia and akoria. The former terms are meant to describe an intense and almost uncontrollable appetite, a sort of canine hunger. This desire for food often leads to the taking of large quantities. although this is not necessarily the case. When the appetite is for quantity and when the hunger is not so intense, the condition is termed polyphagia. There are certain patients who suffer from a continual craving sensation which is not satisfied by the taking of food; in fact they do not have intense hunger and rarely eat in large quantities. Strange as it may seem, they may have little appetite and yet are never satisfied. These various conditions may exist unassociated with any known gastric lesions, and for the most part they are the expression of a neurosis. It is true that bulimia sometimes accompanies hyperchlorhydria and also there is a tradition that any one of the conditions here described may result from the presence of an intestinal parasite. This association has not been noticeable in my experience. Polyphagia may lead to dilatation of the stomach or to gastric atony or both. In moderate intensity, the symptom is observed in victims of diabetes mellitus and those who have undergone prolonged fasts, or in the convalescent from the acute infections; but here it should be regarded as physiological. In one of my cases bulimia occurred in a child in association with vomiting. No sooner was the stomach empty than the cry was raised for more food, and this was eaten with earnivorous avidity.

Treatment.—The successful management of these cases involves a question of education and especially of suggestion. I have seen greatest success from joining to psychic treatment some definite regimen or restorative treatment which in itself contributes to the principle of suggestion and lessens the injury to amour propre.

#### GASTRALGIA

Severe epigastric pain, or gastralgia, is to be looked upon as a symptom, not as a special disease. It probably arises in the solar nerve plexus. It may be occasioned by over-stimulation of the stomach walls as in case of ulcer or intense hyperchlorhydria, although from the latter causes alone it is rare. The pain occasioned by sclerosis of the coronary vessels is located sometimes in the epigastrium, when its real nature may be mistaken. I have met with three remarkable cases and in each within a fortnight there occurred rupture of the cardiac walls at the apex, a sequence to which Osler has referred. Gastralgia may be produced reflexly from disease of the appendix, disease of the gall-bladder or other abdominal affection. So frequently is gastralgia the expression of cholecystitis or cholelithiasis that one comes to look first to that region as the source of the pain. Formerly many cases of gall-bladder trouble were incorrectly treated for gastralgia, an error that of late is less often made. The purest type of gastralgia is that which originates in the spinal cord, the result of early tabes. This form,

the gastric crisis, may be associated with vomiting, yet at times pain alone of the most aggravated character is complained of.

Symptoms and Signs.—The diagnosis of gastralgia can be made only by excluding other forms of abdominal pain, and in most instances the symptom must be referred to a location other than the stomach for its origin. (See Abdominal Pain, page 649.) Little is to be learned by examining the stomach contents except in case of ulcer. Generally there is little or no local tenderness, there is pain and nothing else. When gastralgia is associated with other definite symptoms, these may be so manifestly related to other regions that the location of the pain is of minor importance. Such for instance is the pain that occurs from pancreatitis or pancreatic calculus. When the gastralgia results from tabes there may be lacking most other symptoms of the affection, but we may be set right in diagnosis by the history of syphilis or by positive Wassermann reaction. At one time I supposed that syphilis of the stomach was the direct cause of gastralgia, but I have come to believe that the latter is usually produced through the medium of the spinal cord. It should be remembered that hysteria may counterfeit gastralgia and the latter may also constitute the aura of grand mal.

Prognosis.—Tabetic pain in the stomach may continue paroxysmally for years in spite of treatment. As the disease advances the frequency of the attacks subsides. (See Syphilis of the Stomach, page 620.) When the pain is a symptom of disease of the gall-bladder it is readily controlled by proper treatment, surgical or otherwise. When it results from arterial sclerosis, located either in the heart or the mesenteric vessels, palliation of the attacks is all that can be expected. When it arises as the

result of local irritation of the stomach, the outlook is very hopeful.

Treatment.—Too often the sufferers from persistent gastralgia become victims of the morphin habit. I feel that in some cases this is unnecessary for the reason that the coal tar preparations properly used may be successful in controlling the pain. Very large doses may be required, as aspirin in 1.50 gram doses (gr. xxii) or the equivalent in antipyrin or phenacetin. alone or preferably in combination with hyoscyamin hydrobromate, or sulphate or all these combined sometimes work surprisingly well. Success also follows the use of quinin in large doses. Chromium sulphate in doses of .60 gm. (gr. x) four times a day sometimes succeeds without serious ill effects. In syphilitic cases long continued inunctions of mercury or mercury by injection, or salvarsan or enesol should be tried. For temporary relief, we should use first prolonged irrigation with hot water through the stomach tube followed by the hot Priessnitz pack. Internally, compound spirits of ether, chloroform water, nitroglycerin and valerianic acid are of benefit.

When the result of peptic ulcer or other local lesions of the stomach, gastralgia is readily relieved. Paregoric, or a pill containing 0.06 (gr. i) each of opium, extract of hyoscyamus and camphor, are favorite remedies.

In the course of many diseases the gastric digestion becomes a matter of importance. Although attention has been given to the action of certain diseases in causing functional derangement of the stomach, there yet remains something to be said in special discussion of some of these and of others in their relationship to the primary digestion. The stomach in its relation to tuberculosis and respiratory disease, to cardiac, renal, febrile, arthritic and cutaneous disease is considered in the next chapter.

#### CHAPTER X

THE STOMACH IN RELATION TO OTHER DISEASES

# THE STOMACH IN RELATION TO RESPIRATORY DISEASES

The respiratory tract both upper and lower may suffer as a result of faulty primary digestion, and on the other hand the digestion is often disturbed in sympathy with coryza, tonsillitis, bronchitis, etc. A striking result of pleurisy is the complete anorexia which often attends The gastric disturbance is more than secretory, showing itself in various degrees of atony, temporary in character, usually subsiding before the respiratory trouble has disappeared. Certain individuals after indiscreet eating are likely to experience tonsillitis, nasopharyngitis, bronchitis or asthma. These reactions to indiscretion in eating are sometimes the direct results of over-taxation of the stomach, but more often there is associated an intestinal trouble with headache and metabolic derangement. Often the respiratory symptoms appear promptly after meals, and as digestion progresses the symptoms subside. A common illustration of this is larvngeal irritation and lack of tone in the larynx showing itself in weakness or roughness of voice if one attempts to speak or read aloud after dinner, an experience well known to actors and singers. Occasionally cough is excited and dyspnea may result upon active use of the voice after a hearty meal. It is a mistake to suppose that hearty eating improves a cold. In point of fact a coryza or bronchitis increases in intensity upon taking any except light

200

food, a person with chronic bronchitis finds that his cough and expectoration increase after dining and asthmatics suffer from increased dyspnea or paroxysms of asthma after eating heartily especially in the evening. Apparently gastric stagnation frequently causes reflex bronchial irritation. This may result from excitement of the vagal nerve endings, which would explain the fact that atropin hypodermically acts as a sovereign remedy with certain asthmatics, although quite useless in others. The majority of asthmatics are more certainly relieved by the subcutaneous injection of 1 c.c. of a 1-1000 solution of adrenalin, which according to the conclusion of von Noorden and Eppinger and Hess may be considered as proof that the spasm in these cases of asthma is the result of excitement of the sympathetic nerves. While these therapeutic facts appertain to most cases of asthma, they also apply to asthma having a basis in definite gastric disturbance. These cases are believed to have a digestive cause for the reason that attacks appear especially after the taking of hearty meals, and because the asthma may be aborted by emetics, by gastric lavage, and sometimes by the taking of an active purge like rhubarb and senna. I have known patients convalescing from pneumonia to suffer from severe and even fatal pulmonary congestion and edema after a large and indigestible meal. The general rule may be laid down that a light and digestible diet is the only proper one for most patients suffering from respiratory diseases so that indigestion may not be excited and patients suffering from indigestion must have an easily digestible diet lest respiratory symptoms be induced.

#### ASTHMA DYSPEPTICUM

This term was used by Henoch to describe a rare symptom complex which occurs both in children and adults in

which dyspnea is a prominent symptom. Reigel justly remarked that the name dyspeptic asthma is of doubtful propriety, as its clinical manifestations and physical signs are unlike those of other forms of asthma that have been described. The attacks come on rapidly and consist of greatly accelerated respiration, a rapid, small and perhaps scarcely perceptible pulse, cyanosis, coldness of the extremities, a subnormal temperature and, at times, the complete symptomatology of collapse.

The term dyspeptic is applied to these cases because during the attacks there is usually evidence of distension of the stomach, and the attacks often subside after vomiting has taken place. The exact nature of these attacks is not understood. Henoch attributed them to reflex action, starting in irritation of the gastric mucosa. horn believes that the condition depends on vagal irritation. A number of others would explain the condition as an effect of gastro-intestinal intoxication. All unite in implicating the stomach in the causation. It is a fact that asthma dyspepticum occurs in those suffering from some form of gastric trouble. Achylia gastrica, hyperchlorhydria, chronic gastritis, gastrectasis, and various other affections of the stomach have been found associated with this affection.

The disease sometimes occurs in chronic form, or more properly speaking, certain individuals during a considerable period of time are subject to recurrences. In the ordinary type of the disease the symptoms persist for but a few hours, whereas in the chronic form the symptoms may be longer continued and may recur so long as the stomach is allowed to remain in a disturbed condition.

It is probable that several distinct affections have been classified as asthma dyspepticum. In acute dilatation of

the stomach elsewhere described, there is rapid heart action, cyanosis, symptoms of collapse and sometimes rapid breathing, and this condition also may terminate by vomiting. However, cases of acute gastrectasis which I have seen at no time suggested asthma. Though the breathing is often accelerated, tachypnea, which is characteristic of asthma dyspepticum is not, so far as I know, ever present in acute dilatation.

Acute dilatation of the right heart accompanied by gastric derangement may give the symptoms of dyspeptic asthma. In fact, Silbermann believes that some cases are caused by paralysis of the right heart. The suggestion has recently been made that the attacks are dependent upon a sympatheticotonic condition in which the vagus action is greatly inhibited. It is not easy to explain the affection by means of this hypothesis, yet it is quite as reasonable as the other theories which have been advanced.

Prognosis.—The attacks usually disappear within a few hours, but sometimes persist for a day or two, varying in intensity. They are not devoid of danger; in one of my cases occurring in a child death resulted at the end of eighteen hours, yet nothing was found at autopsy to explain the case.

Treatment.—As has been noted the attack usually subsides after vomiting has occurred. For that reason, it is recommended that the stomach be emptied by lavage. Some benefit might follow stimulation of the vagus by making direct digital pressure in the neck over the course of the nerve. Oil of amber in doses of half a drachm (2 c.c.) administered after lavage is a promising remedy. After the subsidence of active seizure, the stomach should be treated for the relief of such pathological condition as may underlie the disease.

#### THE STOMACH IN TUBERCULOSIS

Although the stomach is largely exempt from tuberculosis, yet dyspeptic symptoms are prominent in the disease, especially when the respiratory tract and the abdomen are involved. An explanation of these symptoms may be found in gastritis, usually of the chronic form; as a rule the symptoms result from functional disturbance or are produced by fever.

Many cases of pulmonary tuberculosis are introduced by a period of irritable stomach; the symptoms may be occasioned by vagal irritation. The clinical picture resembles that which results from chronic irritation of the appendix or the gall-bladder.

Case No. VI. Symptoms of Mild Cholecystitis, Nervous Breakdown; Later, Vagotonic Dyspepsia and Tenderness Over the Gall-bladder; Cholecystostomy; Gall-bladder Found Healthy; Death Two Years Later from Pulmonary Phthisis.—An unmarried woman, aged 24, had suffered a slight attack of jaundice two years before entrance. She developed mild neurasthenic symptoms and resorted to a rest cure. Meantime she experienced much gastric distress with tenderness over the gall-bladder and, occasionally, epigastric pain.

The upper lobe of the right lung showed impairment of vesicular breathing and occasional râles, but there was neither cough nor temperature.

The stomach contents, after an Ewald test breakfast, showed a total acidity of 56, with free HCl of 32, no organic acids and no mucus. There was good motility. As she was but temporarily relieved by treatment, a cholecystostomy was done. The gall-bladder had a normal appearance, although the contents was over-viscid. Brief improvement was noted, yet the patient did not re-

gain good digestive power. Within a year there developed active tuberculosis of which the patient died at the end of two years in spite of approved treatment in well selected climate.

This case illustrates what has been termed "pretubercular dyspepsia"; it also exemplifies an irritative or vagotonic gastric neurosis, easily mistaken for reflex irritation dependent on local disease (cholecystitis, appendicitis). The digestive disturbance of beginning tuberculosis is less amenable to treatment than is dyspepsia having a different origin, yet apparently of the same type. Nearly always the symptoms are those of gastric hyperesthesia, distress after eating, water brash, eructation, anorexia, nausea and ultimately vomiting. Almost invariably there is epigastric tenderness, often a burning sensation or pain.

No explanation of these symptoms is to be found in the gastric chemistry of most cases. Hyperchlorhydria, however, sometimes is present. The use of antacid remedies, the early selection of an easily digested, unstimulating diet, and removal from the sources of fatigue and nervousness produce the best results, yet the condition continues to relapse until the general health is restored.

The gastric manifestations of tuberculosis previous to the appearance of physical signs, correspond to the dyspeptic symptoms which accompany the moderately active stages of the disease.

One of the prime factors in the therapeutics of active tuberculosis is superalimentation, but when this is carried out with a defective digestion it defeats its own purpose; therefore, it is desirable to know the state of the primary digestion. A good many investigations of the stomach contents in such cases have been made but the results are not uniform. Even in cases which are free

from gastritis there occurs in a proportion marked gastric irritability, at times anorexia and vomiting. These symptoms seem to depend partly upon fever, for during that period of the day when the temperature is lowest the patient has appetite, while during the height of the temperature there is complete anorexia. Even when the patient is able to eat during the period of hyperpyrexia gastric distress ensues. In further support of the theory that the gastric disturbance is somewhat dependent upon fever, it is found that after the administration of antipyretics there is improvement in the appetite and in the toleration of food.

Apart from the effect of temperature there are displayed in the actively turberculous the various types of functional gastric derangement. Doubtless they are partly psychic, for tuberculous patients usually exhibit abnormal mental depression at times varied by exhilara-Moreover, the irritability of the nervous system, because of lowered nutrition and the intoxication special to the disease, gives rise to functional derangement of the stomach. There occur anorexia in various degrees, pain after eating, a troublesome sense of repletion, at the beginning of a meal, or other numerous forms of distress that may attend gastric hyperesthesia. Gastric secretion and motion are doubtless influenced by the condition of the buccal cavity; for instance, through dryness of the mouth and resulting difficulty in deglutition, the appetite is impaired. This experience contributes to disturb the psychic state. Disgust excited by the character of the sputum and anxiety caused by the discovery of blood in the sputum add to the unfavorable state of mind. Derangements of motion secretion and sensation may follow these indirect causes.

Studies of the gastric contents in pulmonary phthisis

206

have yielded contradictory results. The secretion of HCl is less active when the temperature is high, but the secretion of ferments seems to be little affected. general condition of the patient has an influence on functional activity. When there is emaciation, anemia and marked toxemia the gastric secretions suffer impairment. Sometimes in tuberculosis, perhaps owing to excitement of the vagus nerves, there is present an active hyperchlorhydria, the gastric digestion then corresponding with that previously described as occurring in the incipient stage of the disease. It is unusual to find the disappearance of secretion in tuberculosis except when there is associated gastritis. The motility of the stomach is sometimes greatly disturbed. This may be secondary to violent coughing when vomiting occurs as a sympathetic manifestation. Occasionally vomiting is excessive and intractable so that alimentation becomes a matter of great difficulty. Belching and water brash are very common symptoms and depend upon motor excitability. One of the most trying gastric conditions is atony; its development is favored by overfeeding in the attempt to obtain a high state of nutrition.

Treatment.—It is important in all tuberculous cases to obtain a favorable psychic state in relation to eating. Too constantly importuning to eat frequently results in spoiling the appetite. On the other hand unless a sufficient amount of food is prescribed many patients will permit themselves unnecessarily to fall into a state of lowered nutrition. It is best to have carefully considered rules for feeding the patient and to insist that these be carried out, yet so far as possible this must be done without exciting antagonism and disgust. Nicety in preparing and serving foods is of great importance. The heartier meals should be taken during the period of

apyrexia and lighter foods during afternoon and evening. The immediate disposal of sputum, the washing of the mouth with some refreshing lotion and the judicious use of condiments and flavors are worthy of attention. Undoubtedly many tuberculous patients suffer from being overfed. To ascertain the amount of food best adapted to a case it is necessary to make gastric analyses to find how promptly the stomach empties itself and to examine the stools to find how well the food is digested. Many times patients are improved by lavage at night, after which the stomach is allowed to rest until morning. enables the tired muscles of the stomach to regain their power of contraction and puts the patient in better condition for full feeding on the following day. Tardiness in gastric motility may be lessened by the use of carbonated waters, ginger ale or suitable carminatives. Hyperesthesia may be relieved by bismuth, cerium, chloroform water and other measures such as are described in the section on gastric hyperesthesia. When the cough persists and causes vomiting it may be necessary to use small doses of codein. When there is an active gastric secretion associated with motor irritability, benefit attends the use of hyoscyamus. The gastric distress may be relieved by an ice bag placed over the epigastrium, a measure that is especially suitable when there is fever.

Tuberculous patients who suffer from gastritis are often fed excessively, which results in local distress, increase of fever and insomnia. Such patients should be treated by soft diet, gastric lavage, local astringents and other remedies indicated for relief of gastritis. It may be possible to observe this rule and yet introduce an adequate number of food units.

## TUBERCULOSIS OF THE STOMACH

Active tubercular lesions in the stomach are unusual, yet they do occur.

There has been much speculation as to the reason for the relative immunity to tuberculosis on the part of the stomach but no satisfactory explanation is offered.

Blumer, who reviewed the history of thirty authentic cases and described a case of his own, classified the lesions under three heads: (1) miliary tuberculosis of the stomach; (2) single tubercular ulcers, and (3) multiple tubercular ulcers of the stomach.

The lesions studied for the most part showed evidence that the infection had been transmitted through the blood stream, less often through the lymphatics. In miliary tuberculosis the stomach may be involved together with other organs but the mucosa is rarely attacked. In exceptional cases the mucosa suffers, probably from direct infection.

At autopsy there are sometimes found unsuspected small tubercular nodules. Lesions may show ulceration or loss of structure, or new formation and hypertrophy. The ulcerative form occurs most often along the lesser curvature near the pylorus where lymphatic tissue is abundant. These lesions are usually single but they may be numerous. They vary from half a millimeter to several millimeters across. Musser 2 reported an instance of gastric tuberculosis in which there was an ulcer about three inches in length by one inch in breadth.

They are round or ovoid, their center hollowed out, and borders undermined, with the overhanging margins thin and unsupported. The floor of the ulcer may show

<sup>&</sup>lt;sup>1</sup> Albany Med. Annals, 1898, p. 145. <sup>2</sup> Phil. Hosp. Reports, 1890.

caseous tissue or granulations; the tissue immediately surrounding it is raised above the plane of the healthy mucosa.

Microscopic examination shows characteristic tubercular tissue. Besides the necrosis of glands there is embryonic infiltration; giant cells occur occasionally. Tubercles in various stages of development are seen and in some of these bacilli are to be found, but these are rarely numerous.

The mucosa surrounding these tubercles shows evidences of gastritis; the lymphatics are enlarged; the peritoneum corresponding with the lesions often exhibits a thickening, the result of inflammation.

Owing to the blocking of vessels by obliterating arteritis and phlebitis, hemorrhage does not often occur.

The proliferation, ulceration and fibroid changes, may produce stenosis of the pylorus or, the ulceration extending deeply, may cause perforation.

# TUBERCULAR PYLORIC STENOSIS

Tubercular pyloric stenosis described by several observers, may depend upon ulceration and contraction or upon interstitial proliferation of tubercle tissue, mostly submucous. These masses or tuberculoma may reach sufficient volume before breaking down to obstruct the pylorus. Or the stenosing process may consist of mixed lesions, some of which show hypertrophy, others ulceration, accompanied by secondary inflammation.

It is believed that fibrous, stenosing gastritis may have a beginning in tuberculosis that is recognized with difficulty.

Symptoms.—There is nothing in the history of tuberculosis of the stomach sufficiently characteristic to make a diagnosis possible previous to operation or to autopsy.

210

When gastric symptoms are prominent in victims of pulmonary phthisis, their occurrence is often attributed to tuberculosis of the stomach, which assumption is rarely justified in fact, for even when gastritis is present it depends upon general causes.

When there is actually tubercular ulceration with gastritis it gives rise to the symptoms of simple gastritis; when there is pyloric stenosis the symptoms are those characteristic of obstruction.

Prognosis.—The history of known cases supports the view that tuberculosis of the stomach develops slowly.

The treatment is symptomatic; in case of pyloric stenosis relief should be attempted by surgery.

# STOMACH IN CARDIAC DISEASE

Reference has been made to the sympathetic disturbances of digestion secondary to disease of the heart, but the relationship between the stomach and the heart requires further consideration. In functional disturbance of the heart and in functional disturbance of the stomach the same causal factors are often at work. In either case must be counted some of the varied neurotoxic or other states which produce motor disturbance both of heart and stomach. However, a disturbed cardiac action often depends upon a definite gastric derangement. When there is stopping of normal gastric peristalsis, with accumulation of gas or other content within the stomach, there is likely to result a sense of cardiac depression with arrhythmia, increased frequency of the heart action and sometimes tachycardia. When there is delay in emptying the stomach, some patients suffer from violent heart action with or without increased frequency. Patients often speak of this as "pounding of the heart," and learn to refer the condition to some indiscretion in eating or to the taking of food too liberally when mentally or physically fatigued. Under similar conditions complaint may be made of marked precordial oppression and a feeling of faintness. This may be accompanied by a feeble heart action, perhaps by coldness or sweating. It is sometimes not easy to determine whether the symptoms arise primarily in disturbed heart action or disturbed digestion, but apparently a functional disturbance of the heart is sometimes responsible for gastric derangement. The victims of paroxysmal tachycardia or arrhythmia commonly complain of indigestion, and this is so not only when there is indiscretion in eating, but when the cardiac derangement has been caused by mental excitement or physical effort. The disturbances of the heart that follow faulty primary digestion are often associated with metabolic defect and it is difficult in many cases to disassociate digestion from metabolism. An improper meal may give rise to indigestion: this may be followed by constipation, hepatic insufficiency and disturbance in metabolism, all of which in a susceptible patient may unfavorably affect cardiac action.

Structural diseases of the heart have a more definite and more easily understood, harmful effect upon digestion. In valvular disease, cardiac dilatation or myocardial weakness from any cause, there is a slowing of the venous current and definite disturbance in gastric secretion and motion results. When there is great cardiac insufficiency the venous congestion may extend to the liver and other abdominal viscera thus contributing to derange the gastric function. Under these circumstances the heart action is made to suffer reciprocally. Every clinician will testify to the ill effects upon the circulatory apparatus of a halting gastric digestion. It is found that

temporary abstinence from food and the administration of a mercurial and saline purge seem to have a more beneficial effect upon the heart than cardiac stimulants or other remedies. Neglect to follow the plain indications of therapy, the continuance of feeding the patient and stimulating his heart when the stomach is inactive, may bring on alarming symptoms or worse. Indeed no little care is required in serious cardiac insufficiency, in order to nourish the patient properly and not to embarrass the action of the heart.

Anginal attacks may be induced by a hearty or indigestible meal with nearly the certainty that is true of mental excitement or undue physical effort. The pain of aneurysm is increased not only by over-eating, but if the stomach is disordered, even by scanty eating.

Paroxysmal tachycardia, when the heart is crippled, may be measurably controlled by assisting the gastric digestion and by demanding no more of the stomach than it can easily perform; the same may be said of arrhythmia, and to some extent these statements apply to auricular fibrillation, extrasystole and partial heart block.

Gastritis readily supervenes when the gastric mucosa is in a state of chronic congestion secondary to valvular or other heart disease; and when gastritis develops in such cases it is hard to overcome. Once established the patient is constantly menaced, for to gastritis the functional disturbances are added, sometimes in spite of skillful management.

## RENAL DISEASES IN RELATION TO THE STOMACH

The functions of the stomach are disturbed and even gastritis is started as a result of intoxications. For instance, a patient, by accident, swallowed a teaspoonful of the fluid extract of digitalis. A friend immediately gave

her castor oil. An hour later the stomach was washed out, but was found empty without a trace of the drug. Three hours afterwards the patient began to vomit; she tasted the digitalis and it could be smelled in the ejecta. Emesis was repeated often during twelve hours; little urine was voided; the heart action was but slightly influenced; however, for some days there were symptoms of gastritis. In this case the digitalis was absorbed from the intestine, it reappeared in the stomach with an excessive gastric secretion and was carried off by vomiting.

This method of elimination occurs in various kinds of intoxications and doubtless is one reason why vomiting is so common a symptom in infectious and toxic diseases. In uremia the stomach assists in the elimination from the blood of toxic material which the kidneys should have removed. The vomiting in uremia is therefore sometimes a vicarious activity and in a sense conservative. Alimentation is prevented, a temporary advantage however; it must soon be resumed. In some cases of acute uremia the patient nearly unremittingly vomits until fatal coma develops. At times the symptoms resemble Asiatic cholera, with vomiting, purging, painful cramp and the algid state. More than once I have seen this mistaken for cholera morbus: once a subcutaneous injection of morphin had been given; the patient fell into a stertorous sleep from which he never awoke.

In mild cases of uremia vomiting may be rare or absent; notwithstanding, the stomach is troubled. Anorexia, gastric unrest, distress and burning are the symptoms complained of, and they continue to occur until rest, with balneotic and other measures of elimination, overcome the intoxication.

Such experiences belong to the history of chronic interstitial nephritis, but identical symptoms arise in renal insufficiency, in which no structural disease of the kidney is demonstrable and in which suitable treatment leads to cure.

Hyperchlorhydria and over-secretion may be caused by kidney disease; lowered secretion and achylia gastrica may originate in a like manner and so may gastritis, gastric atony, gastrectasis, gastric unrest and gastralgia.

The sudden twist of a loose kidney, compressing the ureter and the vessels and the nerves, gives rise to those formidable symptoms called "Dietl's crisis." In a few cases of nephroptosis active functional gastric trouble is produced either reflexly or through vagotonic excitation; so also nephrolithiasis causes stomach symptoms.

Renal disease is affected favorably or unfavorably by the condition of the stomach. The careful diet that forms a leading part in the treatment of kidney diseases may be advisable for other reasons than the metabolic.

An attack of gastric derangement no matter what its nature or cause, may aggravate an existing renal disease. This may come about through excitement or depression of certain divisions of nerves or through metabolic perversions which often follow digestive failure, and which are inimical to the functional activity of diseased kidneys.

## THE STOMACH IN DIABETES

In the early stages of diabetes the primary digestion rarely causes trouble. At first the appetite is increased and there seems to be an unusual activity both in secretion and motion. The active motor function enables the diabetic to empty the stomach even when there is polyphagia and polydipsia. When the disturbance in metabolism leads to marked tissue degeneration, the gastric

functions suffer together with other vital processes. This is not alone due to degenerative changes in the mucosa and the muscular tunic, but also to lowered innervation. Chronic gastritis and lowered gastric motility frequently develop, and their presence appears to affect metabolism unfavorably. The development of gastric disease should be anticipated and if possible guarded against, for its presence is an unfortunate complication Routine examination of the stomach should in diabetes. be practiced. With the advent of ischochymia or gastritis lavage should be practiced, allowing the stomach to be empty a few hours each day thus enabling it to regain its tone. Sawyer of Cleveland looks upon gastric lavage as an important measure in the treatment of diabetes as well as in the relief of the local condition of the stomach. He directs that it be practiced while the patient is lying upon the left side, maintaining that by this expedient the contents is more completely withdrawn.

The functional disorders of digestion in diabetes probably result from innutrition and malnutrition. Gastric derangements occur frequently, but the keen appetite, so often present, may lead the patient to overlook gastric symptoms, and unintentionally to represent his digestion as better than it is.

Besides atony and lowered motility, in many cases there is subacidity or even achylia gastrica. With a deficient secretion there is a tendency to fermentation of gastric contents. The growth of yeast cells, fungi and lactic acid bacilli is favored by the diabetic state.

Dilatation from atony, poor motility, slow emptying of the stomach, low secretion and fermentation may all occur at once.

The common practice of diabetics of drinking large quantities of water results in such a dilution of the gas-

tric juice that digestion is impaired and dilatation favored.

Although reports on the state of the gastric chemistry in diabetes are numerous they are of little value. Each case of diabetes has its individual characteristics. Also, the condition of each patient varies markedly from time to time. It is unwise to take it for granted that the gastric digestion is satisfactory merely because the patient makes little complaint of the stomach.

Occasionally after periods of over-eating or drinking the stomach acts as though it were exhausted, vomiting ensues and sometimes diarrhea. After a short fast the condition passes off and the patient again resumes his dietetic habits. These attacks of gastric intolerance may be an announcement of acidosis; they are not often met with except toward the termination of an unfavorable case, provided the patient is fed in accordance with the indications supplied by careful metabolic study of his case. When acidosis develops, it becomes necessary to administer sodium bicarbonate in large doses which of course antagonizes gastric digestion. Fortunately this alkali is less antagonistic to the digestion of carbohydrates than to proteids, and usually a rather large carbohydrate allowance and a decreased proteid intake is found necessary in acidosis. A knowledge of the fact that sooner or later in a given case resort must be had to alkalies in abundance, should lead to the custom of giving the greatest care to the stomach, so that its functional power may be guarded and so that it may be relied upon in emergency. When it becomes necessary to use alkalies in large quantities, I have found it a good practice to add to the solution of sodium bicarbonate a teaspoonful of milk of magnesia. This considerably overcomes the disagreeable taste and the soda is better

tolerated by the stomach. It is also a good plan to administer bitter tonics, such as the infusion of quassia, condurango or gentian before meals to those who are taking alkalies in large doses.

## THE STOMACH IN FEBRILE CONDITIONS

Loss of appetite and symptoms which are referred to slowness of digestion which are complained of by most patients having fever depend largely upon the systemic effects of the disease. Examinations of the stomach contents in fever patients yield contradictory results and consequently are not always easily interpreted. The secretion may be but little impaired; more often there is a deficiency in hydrochloric acid with less deficiency in the secretion of the ferments. It is difficult to say in a given case how much these results are dependent upon the general condition of the patient, and how much upon special retardation of gastric function. In some instances patients having high temperature and low gastric secretion have been given antipyretics; after the temperature had been depressed thereby the gastric secretion was somewhat higher. It is generally conceded that the mere presence of high temperature diminishes the secretion of hydrochloric acid. Regardless of the state of the secretions, the digestive power of the stomach during fever is always decreased in efficiency. There is a loss in the psychic stimulation of secretion and motion; there is depression of innervation and lowered motility. In the long-continued fevers, such as typhoid, the digestive power is still further weakened because of the cellular changes in the walls of the stomach which interfere with secretion, motion and absorption. Gastric digestion is further hindered through the effect of the disease upon other digestive organs and the organs of elimination.

The treatment of the stomach in such conditions is implicated with the treatment of the case as a whole. is a temptation to allow the patient to make long fasts or to take only the lightest nourishment, but this method often defeats its purpose through the increased weakness which follows. Of late years we are learning to give larger quantities of food, always in the form most easily digested, making the attempt to feed the patient from 2.000 to 3.500 food units in 24 hours. This method of feeding looks to the general improvement of the patient and indirectly to increased power. It may fail sometimes by producing gastric atony. In case there is very poor gastric motility, this may be relieved by giving frequently small portions of carbonated water or a few drops of cognac or some other gastric stimulant. At times, however, a fast of twelve hours is necessary during which only occasional spoonfuls of water may be allowed.

# THE STOMACH IN RELATION TO ARTHRITIS

Many years ago my attention was attracted to an apparently causative relation existing between gastric atony or gastrectasis and arthritis, some evidence of which I presented in a paper read before the Association of American Physicians in 1892. Since then I have observed numerous cases in which symptoms of joint disease have shown exacerbation as the result of stasis either in the stomach or intestines. It is probable that toxins resulting from gastro-intestinal stasis act as irritants to diseased joint structures. Certain people are classed as arthritic. That is, they have joint structures that are unusually sensitive to affronts of any kind, and with them definite arthritic attacks may result from auto-intoxication of a kind that would not affect most people.

I have seen patients having stiffness, pain and swelling in the joints of both upper and lower extremities relieved and in some cases cured by the treatment for atony and food stagnation. Joint trouble depending upon intestinal stasis is more frequent in my experience than that arising from gastric stasis. I have had cases in which both stomach and intestines were concerned; for instance, one had achylia gastrica and atony accompanied by constipation. Occasionally there were intervals of diarrhea, resulting from intestinal indigestion, fermentation and putrefaction of intestinal contents. The fact has often been observed that gout either acute or chronic is made worse by eating, especially when the meals are large and rich in proteids. Of course under such circumstances it would be improper to attribute the fault solely to gastro-intestinal causes. The faulty metabolism must be counted as the general source of trouble, and it is hard to draw the line in these cases between the gastro-intestinal and the metabolic defect.

It should be remembered that in acute local congestion or inflammation, as for instance, phlegmon, there is an exaggeration of symptoms upon the taking of food. This in part is the result of increased blood pressure, in part, perhaps of nerve stimulation, and perhaps from a temporary change in the quality of the blood. Recorded observations tend to show that arthritis deformans and some unclassified forms of arthritis originate from disease in the gastro-intestinal tract. Many of these contributions are not quite convincing and it is evident that the subject demands further investigation before the limitations of gastro-intestinal arthritis can be drawn.

Hypochlorhydria has been regarded as an element in the etiology of "rheumatoid arthritis." Woodwark and Wallis a report the results of gastric analysis in ten typical cases, finding in all a decrease in HCl, free and combined. These authors suggest that active gastric secretion serves as a natural rampart against infection and that infection is more probable in a case of hypochlorhydria.

This may be true, yet experience scarcely justifies the hypothesis that hypothlorhydria is often a contributing factor in the etiology of arthritis.

In the large number of cases of achylia gastrica which I have studied arthritis has been seen but rarely.

Probably there is often confusion between post hoc and propter hoc in regard to this question.

# RELATION BETWEEN SKIN DISEASES AND DIGESTION

Undoubtedly there is an important relationship between the state of the primary digestion and cutaneous diseases, but this relationship is but imperfectly understood. Unfortunately those who are competent to understand and to treat gastro-intestinal diseases are rarely qualified to deal with skin affections; and the dermatologists, although usually willing to admit the interrelation of the pathology of skin and digestion, continue for the most part to treat diseased skin by topical applications and content themselves with prescribing for the supposed digestive derangement various conventional remedies, the use of which should be obsolete and which apparently are authorized only by tradition. There is here an admirable field for the joint study of cases by dermatologists and those who are really qualified to treat diseases of digestion and metabolism.

One of the difficulties in the subject is found in the

<sup>&</sup>lt;sup>8</sup> Lancet, 1912, CLXXXIII, 942.

joining of perverted metabolism to disturbed digestion. In susceptible patients a gastric trouble often begets an intestinal or hepatic complication to be followed by a metabolic disturbance which, especially when there is insufficient elimination, usually terminates in a skin trouble.

Another difficulty is the individual peculiarity of certain patients as to the effect of varieties of food, wholesome to the average man, albeit to some highly disturbing. Such disturbances doubtless are implicated with food anaphylaxis; they often include gastric, intestinal, hepatic, metabolic and cutaneous trouble.

It is because of these difficulties that so little is actually known of the relationship between digestion and skin diseases.

In children, excess in animal proteid frequently excites dermatitis which may become a protracted eczema. Animal lovers will have encountered similar results in a house dog that has been improperly fed. It is probable that faulty digestion gives rise to toxic proteid substances which, passing through the circulation, irritate the integument directly; also it is probable that the same causes overburden the metabolism which results in still further toxemia.

Analogous experiences are to be noticed at various ages, and denote not alone excesses in eating animal proteid—not lobster, crabs, shell fish, etc., well-known offenders—but unfavorable results may also follow the eating of vegetable food, fruit, acid, sugar and other usually inoffensive foods. Not infrequently the fault lies in combinations of substances, delectable to most, but singularly disturbing to others. Thus I have known people who found sugar, butter or eggs to be digestible and wholesome taken alone, but who would suffer from gas-

222

tric distress and urticaria if these articles were taken cooked together as in a cake.

Urticaria is frequently induced by taking unwholesome food and is almost invariably associated with gastric irritation, distress, burning, perhaps by nausea and vomiting, occasionally by diarrhea. These attacks of urticaria are not always caused by food idiosyncrasies; gastric indigestion may be responsible for the attacks and the indigestion may follow the taking of foods that ordinarily are quite suitable to the patient.

The failure of digestion may depend upon fatigue, nervousness or some other transient cause; however, it proves sufficient to bring on urticaria. Sometimes the gastric manifestations and the urticaria seem linked to constipation; indeed, not rarely the symptom-complex starts in the intestines.

Acne is influenced, sometimes to a marked degree, by the diet and the digestion. Particularly is this true of acne rosacea which rarely does well when meats are taken freely. That acne in some way is connected with gastric digestion in certain cases is shown by the good results that are known to follow gastric lavage practiced at night, giving the stomach rest until morning. Needless to say acne is related to several distinct factors; there are to be counted infection, immunity, innervation and intoxication. Nevertheless, just as correction of some genito-urinary fault may lead to a cure in some instances, so correcting a gastric defect will help others. I have known acne to improve coincident with relief of gastric atony, hyperchlorhydria and food stagnation.

Furunculosis during the acute stage is aggravated by eating, especially when the digestion is poor; in individuals who suffer from recurring crops of boils, improvement is noticed when the gastric digestion is corrected.

Psoriasis, often so intractable to treatment, occasionally is markedly benefited by overcoming some coincident gastric disturbance, or by a diet limited in one way or another.

Those who doubt the reality of the effect of the stomach upon the skin may be convinced by observing the flushing of the face, sometimes an erythema, which follows upon the taking of food when there is present a transient ischochymia, over-secretion or gastritis.

Conversely, severe cutaneous irritation, especially when there is pruritus or pain, has a deleterious effect on gastric digestion. Probably this depends upon nervous perturbation, at any rate it results in motor, secretory and sensory disturbance. Sometimes a gastritis appears to owe its origin to cutaneous irritation and infection.

Perhaps the most convincing illustration of the evil effects of cutaneous lesions upon the digestive organs is the appearance of duodenal or gastric ulcer consecutive to extensive superficial burns.

## THE STOMACH IN MALARIA

Accompanying the paroxysms of intermittent fever, anorexia, nausea and vomiting are usual gastric symptoms that approximate those found in other acute infections with fever. However, in intermittent fever the stomach disturbance is at times pronounced; nausea is excessive, vomiting severe and accompanied by violent retching and ejection of duodenal contents, often known as bilious vomiting. Associated with the vomiting, sometimes apparently replacing it, there may be gastralgia. In a proportion of cases the gastric symptoms are intense and more dreaded by the patient than are the other manifestations of the seizure.

In the aestivo-autumnal type of malaria the gastric irritability may be prolonged, adding much to the depression of the patient and interfering seriously with alimentation and medication.

The vomiting, in part, seems to be central in origin; occasionally the periodic gastro-intestinal symptoms largely replace the other symptoms of a malarial paroxysm. In some obscure cases the fever is negligible, the patient complaining only of periodic disturbance of the digestive tract. While the stomach usually gives rise to greater distress, sometimes the intestine is more affected; then there occurs diarrhea, and tormina and colic may accompany the frequent watery dejections.

Malaria is sometimes latent, showing itself merely in digestive derangement. This is more commonly met with in individuals who have resided long in a malarial district and who have either acquired a partial immunity or have restrained the usual paroxysms by continued medication.

While the actual nature of these attacks may be misapprehended in some instances and ascribed to primary stomach trouble, in other instances the symptoms are attributed erroneously to the untoward effects of the necessary drugs.

I have seen examples of very severe periodic gastralgia which could be overcome only by intensive treatment with quinin, when quite unaffected by customary dosage, or when quinin was supposed to be causative of the gastralgia.

Instances of latent malaria are encountered in which the disease betrays its presence by gastric symptoms, often accompanied by the cachexia of malaria, in which the condition may be referred to some other constitutional disease, or to local trouble with the liver or gallbladder. The last mentioned view is made to seem the more reasonable because, after hearty eating, the symptoms become much aggravated. This is frequently noted from the fact of the curious irregularity of the appetite often present in the cases. For days there may be anorexia, and then for two or three meals the appetite may be voracious, almost unappeasable. Following the satisfaction of this unusual hunger the dyspeptic symptoms are sharply renewed.

These digestive manifestations of malaria which seem to depend upon systemic causes are not readily to be distinguished from those which arise from local effects of the disease. It is known that partly from the direct action of the plasmodia upon these organs, partly from the accumulation there of the products of cytolysis, especially hematolysis, the liver and the spleen become congested, irritated and enlarged. The circulation through them is retarded, an inflammatory reaction may develop and function be seriously compromised. Here is found ample cause for disturbance of the stomach. Such disturbance is not limited to that of function; hyperemia is inevitable and gastritis may ensue. When vomiting is entrained, the ejecta contain mucus in excess, are often blood-stained, and actual hematemesis may occur.

Although it may not be possible to do so, it is wise, when practicable, to determine what symptoms depend upon constitutional and what upon local expressions of malaria.

Assistance in determining the state of the case may be had in making physical examination of the liver, spleen, and stomach.

Chronic enlargement of the liver and spleen, with gastritis, the result of malaria, overlooked or but dimly appreciated, are encountered among the ignorant and im-

properly treated in malaria infested regions, but such cases are now less numerous than formerly.

At times the gastric symptoms are secondary to renal lesions caused by malaria. Certain individuals react badly to quinin; in these this alkaloid may occasion severe gastric distress or intolerance, be it from local effects, be it secondarily from cerebral irritation.

It is scarcely necessary to mention the well-known gastric effects of arsenic, so often used in the treatment of malaria in certain places.

Treatment.—The gastric irritability of malarial paroxysms may often be controlled by general sedatives. A hypodermic injection of morphin 0.015 (gr.¼), or codein, 0.03 (gr.½) is often successful. When there are special contra-indications to the use of opiates, relief may attend the rectal administration, in clyster, of chloral, 1.00 (gr.xv) and sodium bromid, 2.00 (gr.xxx), or of antipyrin, 1.00 (gr.xv).

Less powerful remedies are at times more suitable; then considerable relief may be obtained through the use, in very small, oft repeated doses, of wine of antimony .015 (gtt.1/4) in a teaspoonful of water, or apomorphia 0.0003 (gr.1/200) a dose may be given at quarter hour intervals, repeated three or four times.

The ordinary anti-emetics are usually, not invariably, ineffectual; thus bismuth subcarbonate or cerium oxalate may afford relief, especially when gastritis is present.

In aestivo-autumnal fever the irritable stomach may be quieted by the effect of a mercurial saline purge. In some communities there is felt much confidence in the action of the pil. hydrarg. .30 to .60 (gr.v to x) at night, following in the morning with magnesium sulphate 30.00 (5i) in saturated solution, given ice cold. Immediately after this is swallowed, the patient should bite into half

an unpeeled orange, the pungent flavor of which conceals the taste of the saline. Subsequently the stomach may be soothed by sipping the infusion of orange leaves or toast water, by teaspoonful portions of cherry laurel water or perhaps by taking other remedies, such as are described in the article on *vomiting*.

For relieving the gastralgia and other distressing gastric sensations, for overcoming motor disturbances of the stomach in the victims of malaria, no remedy is comparable to quinin. When this alkaloid is not well tolerated it should be given subcutaneously or intramuscularly, sometimes in full doses.

The diet in malaria should be as nutritious as circumstances admit of; in the remittent and continued fevers, alimentation is often difficult. All save the lightest foods may be rejected; yet surprises may await us, for occasionally when the patient has been unable to retain the most delicate food, he will call for and retain some preparation that would seem to be quite unfit.

During convalescence the diet, while abundant and highly nutritious, should not include food that is difficult of digestion.

I recall the dictum of an old practitioner along the Virginia tidewater, that "an indigestible meal would bring on a chill." At any rate the satisfaction of an inordinate appetite occasionally is likely to be succeeded by a paroxysm, in which the gastric and cerebral symptoms are pronounced. This remark applies especially to the severe tertian or double tertian fevers which in some regions develop symptoms of an intensity rarely seen in the malarial patient of the northern states.

## CHAPTER XI

#### PEPTIC ULCER

The mucous membrane and the deeper structure of the stomach may suffer loss of substance from a variety of processes that may properly be called ulceration. This may result from traumatism, corrosives, scalding fluids, from tuberculosis, syphilis or scurvy, or from bacteriological invasion accompanying acute infection. It may appear in the train of cachectic conditions or when the immunity is lost from any cause. While it may be proper to speak of these processes as examples of ulceration, they are not to be considered as necessarily in any way related to peptic ulcer. This latter differs not only in its anatomical appearance, but in the physiological disturbances that accompany it, and it is unsatisfactory to seek the cause of peptic ulcer by attributing it to the processes above described.

Sex.—The matter of sex in the evolution of peptic ulcer assumes less importance the better it is understood. According to the analysis of two hundred cases reported by W. J. Mayo, gastric ulcer occurred in males and females in equal proportion, whereas duodenal ulcer was found in males in 77 per cent and in females in 23 per cent of the cases. This is practically in accord with the experience of other modern surgeons. There can be little doubt of the preponderance of duodenal ulcer in males. The matter may be summed up by saying that gastric ulcer occurs in women somewhat more frequently than in men; duodenal ulcer as well as chronic ulcer

Age.—Ulcer is seen most often in women between the fifteenth and thirty-fifth year, in men between the twenty-fifth and fiftieth year, but it may occur at any age in either sex. The frequency with which it is found in later years may be misleading for the reason that it may have developed early in life and then have recurred or continued in later life with few symptoms. In other words, an ulcer found in a man of sixty may have begun when he was forty. The disease belongs to the middle third of life, but reaches into the first and last thirds. Some cases appear to have arisen in intra-uterine life; duodenal ulcer is sometimes developed in the new-born

also.

Occupation and Geographical Distribution.—On close examination, occupation appears to have little bearing on the occurrence of ulcer. The doubtful statement is made that the Slavs are relatively free from ulcer, while the reverse is true of the inhabitants of Great Britain. It is said that the Japanese and Chinese rarely suffer from ulcer as compared with the people of western nations. Clinically, the majority of cases that are seen in England and Ireland are among young, chlorotic serving women. Little profit arises from the study of the incidence of the disease in small sections of the country, because the possibilities of error are great.

The more specific facts relating to the etiology of peptic ulcer will be considered under pathogenesis.

#### MORRID ANATOMY

Location.—Peptic ulcer selects for its location the mucosa of the stomach, of the terminal portion of the esophagus or of the proximal portion of the duodenum. Exceptionally, after gastrojejunostomy, when the active

gastric juice reaches a more distinct portion of the intestine, the ulcer may develop at that point, that is, in the neighborhood of the inosculation. It is most commonly found at or near the pylorus, more commonly in the stomach than in the duodenum, frequently precisely in the pyloric ring or just beyond it. Within the stomach it selects by preference the lesser curvature and the posterior wall at a point near the pylorus.

#### RECENT PEPTIC ULCER

Recent peptic ulcer begins with loss of structure in the mucosa, its borders sharply defined from the abutting normal tissue. It is usually single (about 80 per cent of cases) sometimes double, rarely multiple. Its size may be that of an inconspicuous point, more often it is that of a dime, and occasionally reaches that of a fifty-cent piece. It is round or ovoid in shape and is most often located near the pylorus in the lesser curve, on the posterior wall of the stomach or in the upper one or two inches of the duodenum. In its progress it severs the blood vessels of the part, which leads to hemorrhage, of more or less importance according to the location of the ulcer and the size of the vessels involved.

In the course of time there appears induration of the borders of the ulcer, either with or without accidental infection, in the efforts towards repair. In recent peptic ulcer these fibrous changes are not noticeable macroscopically; in old ulcer the borders are comparatively hard. The sides of the erosion may be abrupt, giving the so-called "punched-out appearance" of Rokitansky; or they may be slanting from above downward, forming a truncated excavation. In other instances there occurs a terraced descent, suggesting the steps of an amphitheater, and these steps may correspond with the several layers of the

walls of the stomach. At times the wall of one portion is slanting and that of another portion is perpendicular or terraced. The sides of the opening of the recent pep-



FIG. 13.—GASTRIC ULCER. (From Museum, University of Buffalo.)

tic ulcer may be irregularly red, punctate or ecchymotic when viewed at the time of surgical exploration; at postmortem this is less easily made out. In old ulcer the blood staining is not observable except in case of recent opening of some blood vessel. In other words, the discoloration corresponds to the activity of the process. With age, the walls become yellowish, or are indistinguishable in color from the normal mucosa.

The bottom of the ulcer varies in appearance according to the nature of the tissue involved. That is, when it is limited by the muscular layer it looks different than when it reaches to or into or beyond the serous layer, where it is limited by recent inflammatory tissue. Usually it is smooth, occasionally there remain inconspicuous irregularities due to fragments that have resisted digestion.

That portion of the stomach involved in ulcer, viewed from the outside of the stomach, varies in appearance according to the duration of the process and the depth of the ulcer. In recent and deep ulcer fresh inflammatory exudate is seen besides thickening of the serous membrane, which is over-vascular, red and sometimes edematous. Patches of adhering, fibrous exudate may give a rough or tufted appearance. There may be adhesions to surrounding serous surfaces. In the absence of these the roughness, thickening and redness serve to locate the portion over the ulcer. Sometimes it may be recognized by touch when not by sight.

Macroscopic Changes.—The gross anatomical changes of peptic ulcer of the milder type are not altogether known to us. The lesion may be limited to the mucosa, occurring as a small, rather deep erosion, not to be clearly differentiated from erosions of other nature. One of the chief differences is that the lesions ordinarily called erosions are usually multiple, while peptic ulcer as a rule is single. It is probable that superficial peptic ulcer occurs with comparative frequency, accompanied

by no symptoms or merely by those of transient irritability of the stomach, healing promptly and leaving little or no discoverable scar. Even larger and deeper ulcers go on to complete recovery, leaving in the mucosa only a

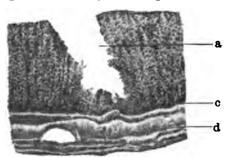


FIG. 14.—GASTRITIS WITH ULCERATION.
(Plate from Text Book of Medicine, Dieulafoy.)

small whitish spot. Still deeper ulcers leave a scar that shows radii of connective tissue forming a stellate appearing depression that is paler than the surrounding mucosa, or is slightly pigmented.

Viewed from outside the serous cover may be marked by a small depression. After recovery from a very deep ulcer there remain the traces of perigastritis with perhaps adhesions.

When peptic ulcer fails to heal quickly, which is occasionally true, when it excites marked round-cell infiltration at its borders so that the healthy mucosa is unable to fold over and, as it were, protect it, then it takes on the characteristics of the chronic peptic ulcer.

## CHRONIC PEPTIC ULCER

We do not know why some ulcers become chronic. The beginning may be undetected because devoid of symptoms and there may be an absence of manifestations until a chronic ulcer is recognized. There is no clinical evidence of stages in its progress and neither post-

mortem information nor surgical exploration afford satisfactory data on this question. It is found that the chronic ulcer varies in distribution and shape as does the recent ulcer, of which it is probably, in the majority of



Fig. 15.—OLD GASTRIC ULCER WITH INDURATION, PERFORATION AND LOCALIZED PERITONITIS WITH ADHESIONS TO ANTERIOR ABDOMINAL WALL. Rod in pylorus. Clinical diagnosis was carcinoma; microscopically shown to be inflammatory induration. History of gastric trouble for ten years.

(Museum University of Buffalo.)

cases, but a continuation. It is usually deeper and often involves the serosa. Through inflammatory changes its border becomes markedly indurated with occasional extension of inflammation into the encircling tissue. Cicatrization and contraction follow prolifera-

tion of granulation tissue, deform the glandular structure, and give rise to a picture that is not always easily distinguished from cancer. Even when the specimen is submitted to microscopic examination, there remains in some instances an opportunity for differences of opinion as to the interpretation. The importance of this differentiation is manifest because we have learned that cancer may begin with ulcer, either replacing it, or forming a mere focus in the cartilage-like ring of scar tissue. (See microscopical section, page 419, and Fig. 20, page 281.)

Chronic ulcer is not a static affair, but becomes at times better and at times worse. The exacerbations may be sudden as when there occurs a deepening of loss of structure, or an extension of inflammation in a new direction, perhaps the result of accidental infection; or when there is a renewal of perigastritis with its special chain of results.

It is because of these fresh extensions of the process that exacerbation of symptoms occurs. Bleeding may arise from erosion of one of the gastric vessels or of one of the hepatic or pancreatic vessels or of the vessels of other adjacent parts into which the ulcer has penetrated; for in the evolution of chronic ulcer, the process may occasionally reach far beyond the stomach. In severe hemorrhage the bleeding vessel is likely to be deep in the ulcer. When mere oozing of blood is encountered, the fault may lie not in the severing of a single vessel but in exaggerated capillary leakage, in which case thrombus formation is prevented because of the very active acid gastric secretion; or the oozing may follow from lack of proper coagulability of the blood. In the course of gastric ulcer, when protracted, there often arise serious consequences resulting from the extensive scar formation which follows efforts toward repair. Often this leads to obstruction at the pylorus. This may come about from thickening, contraction and stenosis (to which clinically is added the element of over-tonicity and spasm). (Radiograms IX and X.) Obstruction may follow perigastritis with adhesions, especially when these anchor the stomach or the duodenum in such a position that there is resulting disturbance of motion or even actual blocking of the channel.

The adhesions often extend to the gall-bladder or its ducts so that there occurs a chain of symptoms due to biliary obstruction and irritation. The small intestines may be encased in adhesions or bound by narrowing bands of tissue that interfere with peristalsis.

Chronic peptic ulcer differs from recent ulcer in appearance. After the abundant development of embryonic elements, the part undergoes a cicatricial transformation which gives a distinguishing macroscopic and microscopic aspect.

The mucous glands and the specific glands of the stomach, as well as the muscle fibers, are atrophied and their arrangement disturbed by the crowding in of round cells and by the fibrous transformation that follows. In the midst of this there are seen areas of recent lymphangitis.

The walls of the blood vessels are atrophied and infiltrated by embryonic cells, definite endarteritis is present. This is sometimes obliterative in character; at other times small aneurysms develop. In so far as the arterial changes lead to obliteration and thrombosis, there is a decrease in the tendency towards hemorrhage. On the other hand, the tissues thus subjected to a diminished blood supply are more vulnerable to the digestive action of the gastric juice. As for the vessels that undergo



Radiogram No. IX.—PYLORIC STENOSIS
FOLLOWING CHRONIC ULCER. There
is marked dilation of the prepyloric
third of the stomach. Note slight escape of bismuth through the pylorus
an hour after meal. Case cured by
posterior gastroenterostomy.



Radiogram No. X.—PYLORIC STENOSIS FROM ULCER CAUSING DILATATION AND STAGNATION. Little bismuth has passed the pylorus. Case cured by pyloroplasty. Pyloric canal found no larger than a wheat straw.



Radiogram No. XI.—PYLORUS DRAGGED DOWNWARD AND TO THE LEFT. Although dilated, the lower portion of the stomach shows marked peristalsis which was visible through the abdominal wall.



Radiogram No. XII.—CICATRICIAL PY-LORIC STENOSIS WITH DILATATION AND BULGING OF THE PREPYLORIC RE-GION, THE PYLORUS THUS BEING COV-ERED OVER.



aneurysmal change, they become an element of danger because when opened they lead to severe though rarely uncontrollable hemorrhage.

The glandular lesions are those of deformity, atrophy and proliferation. Fibrinous infiltration occurs in the glandular tubules, sometimes leading to the formation of retention cysts. The atrophy and the proliferation of the parietal and peptic glands lead to a curious histological appearance. In some areas the over-crowding of epithelial elements give a resemblance to carcinomatous infiltration. This is a matter of importance for the reason that it is in just such areas that, because of intense irritation causing fibrous induration with degeneration and multiplication of the epithelial elements, cancer makes its beginning. It is a remarkable fact that cancerous transformation occurs often in the stomach, though it is rarely seen in the duodenum.

To review these anatomical changes which belong to gastric ulcer, it will be seen that they are readily divided into primary and secondary. The primary changes are not easily explained, and the study of their origin leads us into some of the most fascinating pages of pathology. The secondary changes follow the usual laws of inflammation and repair. Nevertheless, these benign and reparative efforts frequently lead to great functional embarrassment and to the development of important phases in the clinical history of peptic ulcer.

Microscopic Changes.—Since the nature of peptic ulcer involves loss of substance, in part at least, through the digestive action of the gastric juice, to this extent there is little change to be discovered microscopically in the surrounding tissue which makes up the boundaries of the lesion.

There are probably, antecedent to erosion and loss of

substance, certain physiologic derangements and certain structural changes in the part consequent to which there takes place in the focus a loss of immunity, a depression in tissue resistance, so great that it can no longer successfully oppose the attack of the digestive ferments. These changes in the vitality of the tissue are not often recognized and are still open to discussion.

Microscopically peptic ulcer shows: (1) focal necrosis and the effects of autodigestion, and (2) the result of the reaction of repair with round cell infiltration and the formation of scar. Naturally, these secondary changes increase with the age of process. In a very recent ulcer comparatively little microscopic change is to be found, whereas, in chronic ulcer the border and the parts proximate thereto undergo transformation into connective tissue, which varies in extent, according to the intensity of the inflammatory process, its duration and the number of relapses that have occurred.

#### THE EROSIONS

In the evolution of recent ulcer we must conceive of some sort of local disorganization which opens the way for autodigestion. Our actual knowledge of such anatomical beginning is incomplete.

Erosions of Dieulafoy.—Under the term "exulceratio simplex of the stomach," Dieulafoy describes a small erosion involving the mucosa and only rarely penetrating the muscularis mucosa. He states that the clinical history of the affection generally begins with hematemesis, without the usual antecedent history of irritative dyspepsia. Cases which he studied at autopsy and at surgical operations led him to conclude that the process differs from simple ulcer or Cruveilhier's disease. He looks upon it as a form of erosion which is distinctly

hemorrhagic, and recounts one case in which ecchymotic spots were found in other portions of the stomach. Dieulafov's positive statement, added to their own experience, has led certain other observers to support his contention that this represents a special form of gastric ulcer or erosion. It is distinct also from the gastric erosion described by Einhorn, which is elsewhere more fully considered (see page 372). Einhorn's erosion is often associated with chronic stomach symptoms, including distress after eating, a sense of general weakness, loss of weight and the appearance of small fragments of the gastric mucosa in the washwater derived from the fasting stomach. In this form of erosion only slight hemorrhage has been observed, and there is little in the clinical course of the cases to suggest a relationship between the affection and true peptic ulcer. The question of gastric erosion has been widely discussed, especially in France, but the conclusions reached are far from being in accord. Nearly every prominent writer and teacher holds a different view regarding the significance of these lesions. Probably some confusion has risen through the attempt to harmonize recent views with those originally announced by Cruveilhier, who distinguished two varieties of erosions.

Erosions of Cruveilhier.—(1) The follicular, the origin of which Cruveilhier found in the lymphatic follicles and (2) the hemorrhagic erosion. To the first of these he attributed the destructive processes that lead to perforation. This view is not universally accepted. On the contrary, it is held that it is the hemorrhagic erosion, and not the follicular type, that forms an early stage in the development of the round or perforative ulcer, or Cruveilhier's disease.

Erosions According to Hayem.—No author has given

the subject clearer elucidation than Hayem.¹ Not only are his descriptions concise, but his histological studies are convincing. He holds that in reality an erosion of the gastric mucosa represents the second phase of the ulcerous process. Preceding the erosion there occurs a primary lesion, that of focal necrosis. In the destruction

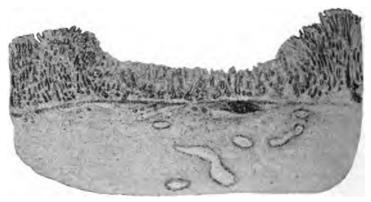


FIG. 16.—SECTION FROM A SIMPLE (SO-CALLED FOLLICULAR), EROSION OF HAYEM. (x18.) There may be observed a distinct, cup-like loss of substance, cutting off the tubules about half their length. There is no eschar. There may be seen under the muscularis, toward the middle of the preparation, an accumulation of lymphoid tissue. Such tissue also exists above and below the muscularis at other points. Some areas show evidence of a mixed gastritis. There is no apparent infiltration of blood nor true congestion; although at the border and in the neighborhood of the excavation there is slight dilatation and stasis in some veins. The submucous tissue is a little swollen and edematous. No proliferation of cellular elements is seen, except in the lymphoid tissue before referred to. The distension of the capillaries and small veins is limited to the zone of the erosion. Some lymph tracts are enlarged. There is evidently slight obstruction to the return circulation in the lymphatics and veins in the area of the lesion. There may be seen minute effusions of blood in the superficial layers of the mucosa between bunches of fibres.

(Plate from "Archives des Maladies de l'appareil digestif et de la Nutrition," page 354, July, 1911. By the kind permission of Prof. Georges Hayem.)

of this necrotic area we find the loss of substance which constitutes an erosion. The necrosis of tissue referred to presents itself in two forms: follicular and hemorrhagic.

<sup>&</sup>lt;sup>1</sup> Arch. des Mal. de l'Appar. Digest. et de la Nut., July, 1911.



Fig. 17.—Section from the Border of Simple Erosion, Shown in Fig. 16. The tubules are filled with altered cells showing beginning multiplication. All of these cells are of the principal type or those derived from this type. Some tubules appear to have ruptured at the edge or in the neighborhood of the neck, allowing to escape cells which are mixed with red blood corpuscles and polynuclear leucocytes. Moreover, there is enlargement and stasis of the capillaries and minute effusions of blood. On the surface is a layer of dense mucus, filled with epithelial cells and leucocytes, which seem to come from the softening of the papillæ and the tissue of the mouths of the tubules, in the interior of which may be seen grayish bits of necrosed tissue together with white and red blood cells. The superficial portions of the tubes exhibit coagulation necrosis. The lesion gives the appearance of having been produced from deposit of a destructive chemical substance on the surface of the mucosa.

(Plate from "Archives des Maladies de l'appareil digestif et de la Nutrition," page 355, July, 1911. By the kind permission of Prof. Georges Hayem.)

Follicular Erosions.—In this variety the necrotic area has a grayish appearance, shows but little blood pigment, and with its destruction there is found merely a superficial erosion of the mucosa corresponding to the follicular type of Cruveilhier. It occurs on the surface of the mucosa as a minute lesion which might readily escape observation owing to the lack of pigment. It varies in size from a minute point to several millimeters in diameter and may not be discovered until the tissues have been prepared by hardening. It resembles a wound caused by digging with the finger nail; at other times its border is abrupt and sharp edged as though cut by a pinking iron. Such erosions are usually multiple; sometimes only a few occurring, at other times large numbers are found.

HEMORRHAGIC EROSIONS.—In the second variety, that of hemorrhagic necrosis, the necrotic tissue is brownish or nearly black, infiltrated with blood after the destruction of which there is found a loss of substance, the tissue still showing blood staining that is characteristic of the hemorrhagic necrosis, and typifies the so-called hemorrhagic erosion. The presence of this type of erosion is announced by the appearance of blood in the contents of stomach and intestines. The question arises as to whether or not the gastric erosions are related, and whether any one of them is sufficient to start the process which we have come to recognize as peptic ulcer; or is there an essential difference between them. Havem believes that only certain types give rise to peptic ulcer. The simple follicular erosion does not in his opinion take part in its development. He regards peptic ulcer as a consequence of hemorrhagic necrosis followed by hemorrhagic erosion and that, in turn, by an eroding ulcer. These conclusions of Havem are better understood by his

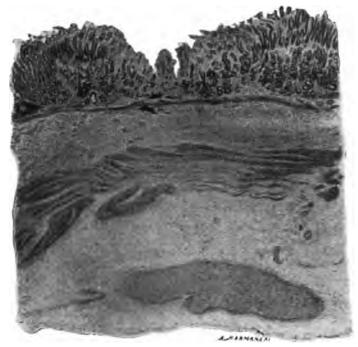


Fig. 18.—Section from a Case of Hemorrhagic Erosion of Hayem. There is general atrophy of the stomach walls. The mucosa shows a mixed gastritis, mostly interstitial. In the center of the depression in the mucosa are two groups of necrosed glands discolored by blood. On the upper and lateral exposure of the sides are little eschars. There is a half necrosed state of the glands as occurs in the "Simple Erosion," but with greater infiltration of blood. The muscularis is intact, but above and below are also accumulations of lymphoid tissue. The submucous tissue is moderately swollen and edematous, closing the somewhat dilated vessels. In the muscular layer is found ingrowing fibrous tissue, scattered and in bands. On the serous surface is attached a shred of omentum, a trace of perigastritis. A large vein is seen, filled with blood, testifying to the stasis existing throughout the diseased area.

(Plate from "Archives des Maladies de l'appareil digestif et de la Nutrition," page 356, July, 1911. By the kind permission of Prof. Georges Hayem.)

illustrations, which are here reproduced (See Figs. 16, 17, 18.)

The presence of hemorrhagic erosions is often announced by the appearance of blood in the contents of

stomach and intestine. Three types described below, are distinguished:

The Lenticular Type.—On gross examination, when the stomach is emptied of clots, one perceives on the surface of the mucosa black or deeply stained spots in points or masses the size of lentils; they are rounded or elongated, and slightly prominent. According to Cruveilhier they are formed by accumulations of coagulated blood, which when removed show superficial erosions, the result of hemorrhagic necrosis. These small excavations have distinct, sharp borders and are circular or ovoid, at times irregular in form. They are rose-colored or black from the imbibition of blood. In number they are usually few, rarely numerous. Their diameter varies from that of the head of a pin to that of a small pea. They occur indifferently in various regions of the stomach. They may be on the side of a ruga, on its summit, or hidden between the folds.

- (2) The Hemorrhagic Infarct.—Hayem agrees with Rindfleisch in considering the hemorrhagic infarct as the cause of a variety of the hemorrhagic erosions of Cruveilhier. It is a focal infiltration of blood, of varying thickness, extent, and shape, measuring in its greatest diameter from 4 or 5 mm. to 2 or 3 cm. One may count in the same stomach several of these infiltrated plaques. They may exist with other forms of erosion. When the specimens are treated by fixation liquid the plaques, infiltrated with blood, tend to detach themselves along the borders as though breaking down.
- (3) Erosions in Furrows.—At times instead of small masses of infiltrated plaques, one observes black streaks of different lengths which detach themselves leaving furrows and fissures. This type of hemorrhagic erosion may be associated with ecchymotic plaques. Invasion by mi-

croörganisms either superficially or through a general infection of the blood may set up inflammatory reaction, cloudy swelling, necrosis and ulceration. These changes are unrelated to arteriosclerosis.

Although arteriosclerosis may cause partial or complete vascular obstruction, necrosis and loss of substance yet it may well be doubted whether these are the usual agencies at work in peptic ulcer. Such pathological processes may be regarded as successful factors in the evolution of a type of peptic ulcer not frequently encountered and not an integral part in the intensely interesting Cruveilhier disease, peptic ulcer. They do enter into the side of the problem now being discussed.

To my mind there is great significance in the finding, by Hayem, and Lion, in the tissue neighboring on very early, typical gastric ulcer, of an edematous infiltration not characterized by evidences of inflammation; that is to say, without abundant embryonic elements. These facts are emphasized by Mathieu in his recent monograph.

In true recent peptic ulcer, the process may extend deeply, even through the serosa. It does not necessarily advance by stages, layer by layer. The glandular structure is cut through, the muscle layer is destroyed and blood vessels are severed; yet these show no arteritis and betray no vestiges of infarction.

Some special form of necrosis, some activity characteristic of peptic ulcer has been at work. There is a devitalization of tissue that suggests, in some respects, that observable in the evolution of the "fat necrosis" of Balser. It appears to take the form of a single area of edematous infiltration, or of hemorrhagic necrosis, or of one succeeded by the other. These constitute what may be called the *primary lesions* of peptic ulcer.

The secondary changes soon manifest themselves and as time goes on increase in importance. Infiltration with embryonic cells is seen in the mucosa, the submucosa, and the muscular layer. The muscle fibers are thinned; there is adenitis in the mucous layer with destruction of epithelial cells near the surface with their active multiplication in the depth of the glands. Aggregations of round cells are scattered throughout. The glands and their ducts are somewhat distorted, the capillaries and arterioles show inflammation. There is, in short, a localized inflammation of the part, an instance of focal gastritis, neither interstitial alone nor parenchymatous, but of a mixed type.

Some have essayed to prove a special form of gastritis in the process, but the evidence is not convincing. The truth seems to be that while some cases show a predominance of an interstitial, others show a predominance of a parenchymatous inflammation. Various structures are involved, some in greater degree than others, and the histologic picture is not uniform. There are reasons for differences of opinion, but it is well at present not to go further than to admit the co-existence of gastritis. This relates to recent ulcer, not alone during the first few days but for some time later. Exactly when ulcer should be called chronic does not admit of sharp demarcation. There is no difficulty with the general classification, but there is a middle ground that must remain debatable.

## PATHOLOGY AND PATHOGENESIS

The question of the evolution of peptic ulcer has of late years greatly excited the interest of clinicians and laboratory workers, but the matter still remains obscure. The various theories advanced, although not easily separated, should have some individual consideration. These are as follows: autodigestion; lowered alkalinity of the blood; trauma; inflammation; lymphangitis and lack of unfolding by the mucosa; bacterial infection; cytotoxic influences; hyperchlorhydria and stasis; internal secretions; arterio-sclerosis and obstruction to the circulation; epigastric hernia; nervous and neurotrophic influences.

Autodigestion.—We are called upon to elucidate the origin of a very special process which occupies a unique position among pathological states. A single round area has lost its resistance to the gastric juice. Autodigestion leads to the loss of structure giving rise to a definite "punched-out" appearance in the mucosa. The ulcer is located near the pylorus, at the lesser curvature, on the posterior wall of the stomach, or in the upper part of the duodenum. How are we to explain such a process? answer would be quickly forthcoming by assuming that the diseased area had from some cause become devitalized. It has long been known that autodigestion of the stomach takes place in sudden death occurring during the process of digestion. In other words, the gastric juice will destroy dead tissue. In order to explain the autodigestion of one small area in the stomach, it is necessary to predicate the devitalization of the part to such an extent that its resistance to digestion, its specific immunity, is lost. Experimentation has shown that there is inherent in the gastric mucosa a resistance to the digestive action of its own secretion. The stomach resists not merely because it is alive, but because of the fact that, being alive, it possesses a characteristic and specific immunity to the action of the gastric enzymes and HCl. was shown by the experiments of Claude Bernard that mere living tissues are not entirely resistant to the digestive action of the gastric juice. He introduced and secured in the stomach of a dog the leg of a living frog, and found that while it showed greater resistance than dead tissue, it was nevertheless slowly destroyed by the gastric enzymes. This experiment shows that something more than vitality of tissue must be necessary in order that the mucosa of the stomach shall resist its own secretion. Not only must there be vitality, but there must be also an integrity of function in these tissues, and this would logically lead us to the conclusion that there is something in the function of the gastric mucosa by which it provides itself with a specific-resisting power and with a special immunity to gastric juice.

Alkalinity of the Blood.—At this date it seems unnecessary to consider the theory advanced by Pavy that alkalinity of the blood is the source of protection against gastric juice, for Riegel showed that the gastric mucosa was not acid merely on its surface, but was acid throughout its depth. The frequent concurrence of chlorosis and other forms of anemia with gastric ulcer at one time lent some favor to Pavy's theory as to the influence of lowered alkalinity of the blood. It is highly probable that chlorosis does predispose to gastric ulcer, but that this is owing to the lessening of the alkalinity of the blood is incredible. Unquestionably, if anemia does predispose to gastric ulcer, this effect must be owing to the lowering of immunity in an area of the gastric mucosa through some influence which we do not as yet entirely understand, but it is something far more profound and richer in meaning than the mere question of acidity and alkalinity. If the question required a further confutation, it might well be found in the fact that the gastric juice is not invariably acid in case of peptic ulcer. In a considerable proportion of cases the acidity is not raised and in exceptional instances, even of grave ulcer of the stomach, there has been found a state of gastric anacidity.

Trauma.—It was long believed that trauma of the stomach, pressure, laceration, puncture, etc., were the factors that served to weaken the mucosa at the point where the gastric ulcer developed. It is difficult to understand how this belief could have taken root in view of the observations of William Beaumont in his classical studies upon Alexis St. Martin. It is to be regretted that all experimental work upon the stomach has not been conducted with the singleness of purpose and the perfection of induction which was so admirably displayed by our great countryman. It was found that the stomach was resistant to trauma, and that repair was complete in a surprisingly short time. Since then in many experiments upon animals in which the gastric mucosa has been burned, punched, cut and torn, the mucosa has been observed to close over the wound almost immediately and the lesion to disappear without any tendency towards ulceration.

Cohnheim and Matthes showed that in animals the healing of injuries in the gastric mucous membrane is more rapid than in other regions of the body. This is owing, perhaps, to the remarkable vascularity of the stomach and also in part to the power which the mucosa has of contracting and thus protecting and partially covering over of the wound, as was shown by Schmidt. If traumatic injury of the lining of the stomach is capable of exciting peptic ulcer, it should be found in those cases where abrasion and laceration of the mucosa continue for a long time. That such is not true is illustrated in the history of the following case.

Case No. VII.—In consultation with Dr. Gaylord, I studied a patient who was in the habit of swallowing

knives, broken glass, steel nails, bits of coal and other extraordinary articles. The gastric secretion was not particularly disturbed. As he suffered some inconvenience, a gastrotomy was done, whereupon there were found knife blades penetrating the walls of the stomach; many sharp fragments had to be extricated, some hundred bits of steel, glass, broken needles, etc., were found free in the stomach. There existed a few erosions, but none that resembled true ulcer. The foreign bodies were removed and, after recovering from the operation, the man's digestion seemed no different from what it was before, although he was free from the uneasiness which these bodies, foreign to the stomach, had created.

There are recorded a number of similar observations. There is no reason to believe that local trauma excites gastric ulcer. Possibly there is more evidence that blows over the epigastrium lead to ulcer, but the bearing is dubious.

It should be strongly emphasized, if we are to understand the subject aright, that the stomach possesses an inherent and powerful resistance to accidental injury from ordinary causes. It does not readily succumb to infectious foods nor to wounds from foreign bodies.

It is interesting to note how the lower animals gorge themselves with putrefied animal tissue, fragments of bone and irritating substances without apparent injury to the gastric mucosa, although in the case of dogs the gastric acidity is extremely high.

Inflammation.—Cruveilhier supposed that peptic ulcer was in some way the result of gastritis, yet his critical mind perceived that there was a difficulty in this view, as we may believe from these lines which I translate: "The history of the causes of simple ulcer of the stomach

is enveloped in profound obscurity, or rather this disease admits of all the causes of gastritis. But why should a single point of the stomach be profoundly affected, all the other points of the organ being left in a state of perfect integrity? Indeed this would seem very difficult to explain." It is astonishing to find how thoroughly Cruveil-hier comprehended the nature of the disease which he was the first to describe. Little of importance has been added to our knowledge of peptic ulcer since his time, and that little has come to us recently, since we have learned to understand more of the principles of immunity operating in health and in disease.

Lymphangitis and Lack of Infolding by the Mucosa.—
The views of Cruveilhier have been reawakened from time to time and recently Galliard has lent his support to the doctrine. While it is incontestable that inflammation accompanies peptic ulcer, this probably is a secondary manifestation in nearly every instance. When as a result of arteritis and lymphangitis the nutrition of the tissues forming the borders of an ulcer is depressed, it is easy to understand that digestion of the part would make headway and that the ulcer thereby would extend; but this merely shows the steps through which an ulcer becomes indurated and chronic and throws no light upon its beginning.

Dolbey 2 basing his views on the studies of C. H. Miller 3 whose work he personally watched, emphasizes the importance of lymphangitis in gastric ulcer. In one case described by Miller the vessels of the submucosa were much degenerated, the adventitia infiltrated, the media thickened, and the intima showed such proliferation that the lumen was in places obliterated by ragged masses

<sup>&</sup>lt;sup>2</sup> Bitot and Papin in France; Dal Lago in Italy.

<sup>&</sup>lt;sup>3</sup> "The Histology of Gastric Ulcer," Arch. of the Path. Inst., London Hospital, 1906, Vol. I.

of clot adhering to the wall. The ulcer was shallow. Dolbey quotes Matthes on the redundancy of the gastric mucosa and the folding over of the mucous membrane, to cover and protect lesions of the part. When this contraction of the mucosa was prevented by sewing in a metal ring, the mucosa thus embarrassed underwent digestion. The author then proceeds to show that Miller found that lymphoid tissue exists about the pylorus and the lesser curvature of the stomach far in excess of that found in other areas of the stomach. He believes that this lymphoid tissue becomes infected and breaks down, the vessels of the mucosa become diseased, the nutrition of the part is decreased and the indurated mucosa loses its power to make protecting folds. The author suggests that disease of the lymphoid tissue is an essential cause of gastric ulcer and that its abundant location near the pylorus explains the frequent occurrence of ulcer at that point. The accompanying vascular disease and rigidity of the mucosa he regards as contributing factors. He furthermore believes that great induration often occurs at the pylorus as a result of these conditions, and that such indurations are often regarded as carcinomatous, when in fact they are entirely benign. He alludes to the tonsils and other lymphoid tissues of the throat and to the extensive distribution of lymphoid tissue in the ileum near the ileocecal valve as analogous to the massing of lymphoid tissue near the pylorus.

These views lead us to recall the teaching of Cruveilhier as to erosions resulting from necrosis of the lymphoid follicles already considered. In reference to the protective effect produced by the folding over of the gastric mucosa referred to by Dolbey,<sup>4</sup> it is well to remember

<sup>&</sup>lt;sup>4</sup> "Gastric Ulcer," Surg., Gyn. and Obst., R. V. Dolbey, M.S., London, F.R.C.S., Eng., Vancouver, British Col., Sept., 1909.

the conclusions reached by Block, that the contraction of the walls and the folding over of the gastric mucosa take place in a very limited degree in that region of the mucosa between the pars pylorica and the lesser curvature, the region in which ulcer of the stomach is frequently encountered. An injury made to this portion of the stomach scarcely can be covered, while injuries in other portions of the stomach are quickly concealed by this folding over process and hence are relatively guarded from the destructive action of the gastric juice.

Bacterial Infection.—The question of gastritis and especially of lymphangitis in relation to ulcer is incomplete without reference to bacterial invasion. In a series of experiments upon animals, F. B. Turck 5 reached the conclusion that bacteria played an important part in the formation of gastric ulcer. He succeeded in exciting gastric and duodenal ulcer in animals, the resisting power of which had been reduced by starvation and through feeding to them enormous numbers of colon bacilli. the other hand Mathieu says that M. Bauer reports in a yet unpublished account of control experiments that he was not successful in reproducing ulceration of the gastric mucosa analogous to true ulcer. Block concludes from his investigation that bacteria play scarcely any part in the development of peptic ulcer. The microörganisms that are found post-mortem are to be regarded, he says, as finding their way there after death. holic, so prone to gastritis, have not in my experience been specially prone to gastric ulcer, although I admit that this is contrary to the experience of others.

The coincidence of definite infection with peptic ulcer is met with sufficiently often to lead us to believe that there exists more than an accidental relationship. This

<sup>&</sup>lt;sup>5</sup> Int. Med. Congr., Lisbon, 1906.

is well illustrated in the case recounted by Descoeudres.6 A man of 30, for fifteen years alcoholic, had for thirteen years suffered from symptoms of ulcer of the stomach, including gastralgia and hematemesis. Gastro-enterostomy was required to give temporary relief. An indurated ulcer was found. In spite of great prudence in eating, the trouble recurred. Meantime it was found that there was slight but continuous swelling about the submaxillary gland, with the discharge of small quantities of pus through the canal of Wharton. It was discovered that this trouble appeared coincidently with the gastric trouble. An X-ray examination revealed a shadow in the gland. Ultimately the gland was removed, including a salivary calculus. This led to the cure of the mouth infection, whereupon there was complete disappearance of the evidences of ulcer.

Another case is related, that of a shipwrecked sailor who subsisted sixteen days upon putrid bread and water, whereupon a gastric ulcer developed. Mayo Robson and Monyhan hold that infection of the buccal mucous membrane is the cause of many cases of gastric ulcer. Personally, I have seen several cases of erosion and gastritis coincident with periodontitis, but I have never been able to satisfy myself that there existed a relationship of cause and effect between any case of mouth infection and gastric ulcer coming under my immediate observation. When we recall the relationship that exists between the various general infections for instance pneumococcus, staphylococcus and malaria, and herpes, affecting not only the face and lips but the buccal mucous membrane, it leads one to suspect that there may occur some indirect result from infection that leads to the formation of gastric ulcer.

<sup>6</sup> Bull. Méd., Paris, March 6, 1907.

Cytotoxic Influences.—Rosenau and Anderson, of the U. S. Hygienic Laboratory, report having successfully produced gastric ulcer in guinea pigs by the injection of diphtheria toxin. Sellers of Johns Hopkins, experimenting with bile salts introduced into the peritoneum of guinea pigs has produced gastric ulcer. He suggests that in the presence of bile salts the serum no longer protects the stomach against its own ferments.

A specific gastrotoxic serum has been described by C. Bolton.<sup>8</sup> This is said to contain an agent that produces necrosis of the lining of the stomach and another that excites hemorrhage in the same part. It is probable that we shall soon possess further knowledge that will illuminate the obscure field of the native immunity of the stomach against its own secretion and of the various agents that, under certain conditions, may nullify this immunity and perhaps produce the local edema and hemorrhagic necrosis which are admitted to be initial steps in the evolution of gastric ulcer of the usual type.

Hyperchlorhydria and Stasis.—Just what relationship exists between hyperchlorhydria and peptic ulcer is a disputed point. Ewald considers the ulcer to be the source of the irritation that induces over-secretion and over-acidity. Riegel on the other hand was of the opinion that the hyperchlorhydria usually antedated the ulcer. He held that certain individuals having an irritable nervous organization were predisposed to hyperchlorhydria upon trivial cause, and that local injuries to the stomach, which in most individuals were without consequence, might in the more susceptible of these give rise to hemorrhagic erosions and the exaggeration of an already present hyperchlorhydria, and consequently to the digestion

<sup>&</sup>lt;sup>7</sup> Arch. for Int. Med., Nov., 1909. <sup>8</sup> Trans. Royal Soc., Sept. 28, 1904.

of the eroded structure and to the further extension of the ulcerative process. Paul Cohnheim on the contrary says: "Hyperchlorhydria, as such, never causes peptic ulcer," and finds support for his view in these lines which he quotes from Rosenheim: "The predisposing causal factor of ulceration of the stomach is a local reduction in the resistance of the walls of the stomach caused by some disturbance in the circulation that weakens the resistance of the stomach wall against the digestive power of the gastric juice."

For my own part, I find insufficient support for the doctrine that hyperchlorhydria is a necessary phase in the evolution of peptic ulcer. In fact it is known that in about 33 per cent of all cases hyperchlorhydria is absent even after the ulcer has developed. When it is present it undoubtedly contributes to the further extension of the ulcer because it heightens the irritability of the stomach, increases the element of spasm, and hence leads to stasis of the acid gastric juice. Besides this the digestion of the devitalized focus goes on more rapidly with an over-acid gastric secretion. The general statement may be made that in proportion to the degree of hyperacidity the ulcer is rebellious to treatment, and liable to become chronic. It may be safely affirmed that hyperchlorhydria and hypersecretion are contributing factors to, but not the essential causes of gastric ulcer.

The effect of stasis is undoubtedly important. To this Mathieu has recently called renewed attention. Furthermore, it is in relation to stasis that we see most clearly the importance of the acid gastric juice, and for reasons shown in the following facts. The lower part of the duodenum or the jejunum are not under ordinary conditions the seat of peptic ulcer. When, however, a gastroenterostomy is performed and, as a result, the acid gastric

secretion enters directly into the jejunum, then peptic ulcer may develop at this point. In fact the jejunum in respect to ulcer then behaves like the extreme upper part of the duodenum previous to gastro-enterostomy. Of course this does not show that the acidity of the gastric juice is the sole cause of the peptic ulcer, but it seems to demonstrate that the action of the undiluted gastric secretion upon a previously immune portion of the gut was accessory to the evolution of a peptic ulcer in that part. This being so in the jejunum, it is highly probable that it is true of the pylorus and other parts of the stomach especially in case of ischochymia.

Besides this, it is widely admitted that gastro-enterostomy is followed by relief of symptoms and often by the healing of pyloric ulcer or duodenal ulcer located in its usual site, just beyond the pyloric ring. Why this relief of symptoms? Why this healing? Partly because the gastric juice now lacks the opportunity to attack the ulcer focus. A wider explanation may be couched in the general term, rest. For clearness we should know in just what this "rest" consists. Doubtless in part it means a decrease in over-tonicity and over-contractility; in other words, spasm is lessened or overcome. But we must yet explain this effect on spasm. Can this be from any other reason than that the gastric secretion flowing through the new passage no longer reaches, no longer irritates the ulcer in the pyloric region? Evidently the answer must be given with some reservation, but we must admit some importance to the part played by the gastric secretion.

Internal Secretions.—May peptic ulcer depend on some derangement of the internal secretions? There probably occurs a deficiency in antipepsin preliminary to ulcer. The query arises, how is the gastric mucosa pro-

vided with this anti-body in amount beyond that supplied to other tissue? We do not know; apparently it is produced in the part by a special mechanism. It is not the internal secretions alone that enter the circulation; pepsin as well as other ferments is in part absorbed by the blood and may be recovered in the urine. A disturbance of internal secretion, something that would inhibit the focal production of antipepsin and perhaps give rise to cytotoxic substances, is not beyond possibility.

The experimental work of Loeper <sup>9</sup> is suggestive in this connection. He found that the intravenous injection of gastric juice led to exaggerated gastric secretion; also, that the intravenous injection of macerated gastric mucosa produced cytolysis in the lining of the stomach, typical peptic ulcer and perforation with the discharge of fluid markedly proteolytic in action.

The tissues showed no signs of inflammation. These experimental results, like similar findings by others, are not of course conclusive as to the cause of peptic ulcer, but unquestionably they hint at its pathogeny.

Arteriosclerosis and Obstruction to the Circulation.—Virchow long ago called attention to the fact that thrombosis of vessels in the gastric mucosa, leading to hemorrhagic necrosis, would in the presence of active gastric juice result in a peptic ulcer of the stomach. This has subsequently been shown at numerous autopsies. Of course, this accounts for only a small proportion of all gastric ulcers. Arteriosclerosis, however, is by some held responsible for another form of gastric ulcer, and the subject is undergoing discussion in recent literature. I refer to a condition in which there is practically obliteration of a comparatively large branch of one of the vessels of the stomach so that inosculation is made impossible

<sup>&</sup>lt;sup>9</sup> Leçons de Pathologic Digestive, Paris, 1911.

and a relatively large tract of the gastric mucosa is deprived of arterial blood. This condition must be rare, for its occurrence is denied by some experienced clinicians. Riegel referred to it, but doubted its existence.



FIG. 19.—GASTRIC ULCER,

Marked arteriosclerosis, mouths of three arteries showing in the ulcer.

The indurated area about ulcer shows mesh-work of towel which became fixed. (Dr. Williams' Case.)

This was my own view until I saw the evidence presented by Dr. H. U. Williams at a recent meeting of the Buffalo Academy of Medicine. The patient, a woman, fortyeight years old, died at the Erie County Hospital of what was presumed to be carcinoma of the stomach. At autopsy, marked arteriosclerosis was found, aneurysm of the arch of the aorta, and as a result of the obliterating arteritis, the blood supply to a considerable area of the gastric mucosa was interrupted. In the center of this area was a large gastric ulcer from 5 to 8 cm. in diameter, irregular in form but having the characteristic punched-out appearance. This is one of the largest gastric ulcers that I have ever seen. From careful examination of the specimen I am convinced that it resulted from the cause above described, and it may be possible that the very large ulcers of the stomach, occasionally observed past middle life result from this condition of arterial disease more often than is generally supposed.

It cannot be denied that obliterating endarteritis, arterial lesions secondary to endocarditis and thrombophlebitis may be followed by peptic ulcer, provided the gastric enzymes are operative.

From time to time, as a result of a few strikingly illustrative cases found at necropsy, announcement is made by some observer that the essential cause of peptic ulcer is vascular obstruction.

One of the most notable recent contributions of this kind is by Ophüls 10 of San Francisco. He reports a few interesting cases with histologic sections showing an undoubted relation between arterial lesions and ulcer; he affirms that chronic ulcer depends on arteriosclerosis and recent ulcer upon endarteritis. No one doubts that obstruction of a terminal vessel supplying the gastric mucosa will lead to peptic ulcer; but the great majority of pathologists and clinicians are unable to find this cause save in isolated cases.

This view is supported by later investigators more uni-

<sup>10</sup> Arch. of Int. Med., May, 1913.

formly than by earlier pathologists. Aschoff, for instance, whose recent article is quoted by Ophüls, definitely states that ulcer does not depend on primary vascular disease.<sup>11</sup>

Several cases of local arteriosclerosis of the stomach have been reported recently in which no ulcer had developed but in which death resulted from hematemesis.

Sections of the diseased tissue from these cases show the presence of endarteritis with fibrinous and hyalin degenerative changes, obliterating thrombosis of minute vessels and miliary aneurysms.

It is sufficiently established that these morbid changes may produce hemorrhage without the formation of ulcer, ulcer without the occurrence of hemorrhage or both ulcer and hemorrhage.

Epigastric Hernia.—Soper 12 reports his observations on a curious case. A woman 44 years old, who had for a long time suffered from digestive troubles, finally developed severe pain accompanied by vomiting. She had also a very painful epigastric hernia which was reduced with difficulty. The case came to operation, an ulcer was found and closed, and the epigastric hernia was operated upon. Since then the woman has been free from all This observation by Soper is reinforced by symptoms. the report of Ury who also has drawn attention to a relationship between epigastric hernia and ulcer of the stomach. He has reported two cases of his own and recalls three other cases observed at the polyclinic of Boas during a course of ten years, from 1899 to 1909. A case reported by Aaron and referred to in his book is interesting in this connection. I have met with two cases of epigastric hernia accompanied by symptoms of peptic

<sup>&</sup>lt;sup>11</sup> "Aber keine primären Gefässerkrankungen heranzuziehen sind," Deutsch. Med. Woch., 1912, xxxviii, p. 494. 12 N. Y. Med. Jour., Aug., 1910.

ulcer. It seems possible that in all these cases epigastric hernia had influence in leading to the development and evolution of gastric ulcer. One could hardly imagine a relationship between the two processes except through the intervention of the nervous system.

Nervous and Neurotrophic Influences.—Giles de la Tourette held that peptic ulcer is apt to occur in the hysterical and considered it related to the perforative ulcer of the neurotic. There are certainly some very curious cases of this kind like the following.

Case No. VIII.—For a number of years, through the kindness of Dr. Bingham, I studied the case of a middleaged laundress, the victim of hysteria, who from time to time suffered from subcutaneous hemorrhage occurring in well-defined areas scattered over the body especially over the neck and breast. These hemorrhagic areas were irregularly rounded in form, brilliantly red and even turgid in appearance. They were painful and tender to touch. They developed suddenly after a few hours of pain, so that she was able to foretell the coming and the location of the hemorrhage. During some of these attacks, or sometimes at other periods, she suffered from gastric ulcer or at least she exhibited the classical symptoms of the disease, including gastralgia, strictly localized tenderness, acid vomiting, and profuse hema-The symptoms were relieved by the ordinary treatment for gastric ulcer. It should be added that this patient had also chronic tuberculosis. There was a scanty mucopurulent expectoration, containing enormous numbers of tubercle bacilli. In spite of these several afflictions, the woman's general health showed surprisingly little impairment, and she insisted upon continuing her work as a laundress. This case is analogous to those reported by Osler.

The ensuing case illustrates a different relation of the nervous system and ulcer.

Case No. IX.—A middle-aged man had for years suffered from perforative ulcer, apparently Morvan's disease, occurring usually upon the thigh. The attacks began with intense neuralgia which heralded the appearance of the ulcer and which continued until the ulcer was healed. These attacks sometimes lasted for months. On one occasion, the patient came to me complaining of symptoms of gastric ulcer. The localized pain was incessant and there was moderate hematemesis. The ultimate history of the case cannot be given as the patient disappeared from observation.

The pathology of so-called dermatitis multiformis is not very clear. In some obscure way it seems to be related to the nervous system. The following case interested me particularly because of the accompanying gastro-intestinal disturbance.

Case No. X.—The patient was a woman 30 years old, the mother of three children. She came from a highly neurotic family. Her health was usually good. Without premonition, she suffered a grave attack of dermatitis multiformis. Not only was the integument generally affected, but the lesions appeared also in the mouth, throat, vulva and rectum. We had reason to suppose that the esophagus was attacked. She developed shortly intense gastric irritability and suffered from anorexia, localized pain and tenderness, nausea and vomiting of thin blood-stained mucus. In short, she displayed a group of symptoms characteristic of peptic ulcer. These stomach symptoms continued some time after recovery from the cutaneous lesions.

Again, there is something most suggestive in that extraordinary disease known as idiopathic hematoma auris,

which is seen not infrequently in the insane, which invariably appears in the concha, rapidly reaches maturity, and disappears only after destruction of tissue and marked cicatricial deformity. It is interesting to note that the affection has been seen occasionally in those having no mental disease or other known disability.<sup>13</sup> paper on this subject, Dr. H. G. Matzinger refers to the complex nerve supply of the ear, with especial reference to the sympathetics, and concludes that otheratoma is a neuropathic affection seen generally, but not invariably, in those suffering from central nerve disease, in which the sympathetic system is seriously involved. might not some similar process have for its local expression the posterior wall, or lesser curvature of the stomach near the pylorus, than which no part of the economy has a more complicated and involved innervation? One is led also to think of the striking manifestations in that curious affection, Raynaud's disease, with its predilection for the fingers and toes, with its advancing steps of syncope, asphyxia and necrosis of the parts.

It is not impossible that asphyxia of certain spots of the gastric mucosa may occur from analogous causes, whatever they may be, and the tissue thus put to a disadvantage would be well calculated to suffer erosion from the active gastric juice.

Superficial burns undoubtedly excite duodenal ulcer. The reason is unknown. Trophic disturbances, resulting from lesions of the central and sympathetic nervous system are held to be a direct cause of peptic ulcer. The experiments of Ebstein upon the brain, and of Schiff, Ewald and others upon the spinal cord were followed by focal hemorrhage and later by ulceration of the gastric mucosa. R. de la Vedora, in a long series of experiments,

<sup>18</sup> Sparling: Med. Rec., Nov. 9, 1891.

injured the celiac plexus and the splanchnics which resulted in producing typical round ulcer of the stomach. Injuries of the pneumogastric were not followed by lesions of the stomach except when they were made below the diaphragm. When abdominal fibers of the vagi were injured, as shown by Yzeran, almost uniformly there developed gastric ulcer, rather typical in character, with death from hemorrhage some months after the experiments.

The presence of a neuropathic factor in the genesis of peptic ulcer, for many years I have maintained to be indicated by established data, which view, on publication, led to adverse comment, notably by Riegel; 14 who assembled against it all the usual reasons for doubt.

At present, especially since prominence has been given to the results of continued disturbance of the autonomic nerves, many observers are including the neuropathic source of the disease. For instance, Rössle,15 affirms this and holds that ulcer is not caused by spasm of the vessels directly, but by indirect obstruction of terminal arterial branches occasioned by spasm of the fibers of the muscularis mucosae producing minute infarctions. is also the opinion of Lichtenbelt. The minute hemorrhagic erosions are followed by autolysis. Bergman believes that the lesser curve is more often involved because that region alone is exempt from rhythmic peristalsis and other gastric movements which, through occasional stretching of the tunics of the stomach prevent stasis in arterioles. 16 It is probable that vagotonic influences are related to the genesis of peptic ulcer; indeed, a tendency is now observed to class ulcer as a mere vago-

<sup>14 &</sup>quot;Diseases of the Stomach," Nothnagel's "Encyclopedia of Practice of Medicine," American edition, pp. 568-569.

15 Münch. Med. Woch., June 18, 1912.

<sup>16</sup> Arch. des Mal. de l'Appar., Digest. et de la Nut., May, 1913.

tonic manifestation. Thus Bergman <sup>17</sup> found evidence of visceral nervous disturbance, presumably vagotonic in 58 out of 60 cases. To adopt this view unreservedly is going too fast; it should be recalled that already Eppinger and Hess report having found typical ulcer both in vagotonic and sympatheticotonic patients.

Recently Westphal has succeeded in producing in animals the formation of ulcers by the hypodermic injection of pilocarpin and physostigmin. The practical inference may be drawn from this that atropin, which inhibits the vagus, should occupy a more important place than it hitherto has done in the treatment of ulcer and in the prevention of its development when there exists local irritation (appendicitis, cholecystitis) which reflexly might cause vagal irritation and spasm.

Without going more minutely into this subject, enough is presented to show that there are seeming contradictions. Why is it that the stomach so well endowed with the power of restoration to the normal should lose its resistance in small areas, especially located at the lesser curvature, near the pylorus, or in the upper part of the duodenum? It is universally admitted that when the tissues of the stomach lose their vitality, autodigestion ensues; in other words, peptic ulcers are formed. It is known that the gastric juice attacks unfavorably living tissues otherwise than that of the stomach, for instance, the leg of a living frog, the ear of a living rabbit. Dead tissue is digested more quickly than living tissue, but other living tissue is attacked more readily than the tissue of the gastric mucosa. Unquestionably there is something peculiar to the stomach by means of which it is protected from the gastric juice. Also there must be some factor that disturbs or destroys the inherent pro-

<sup>17</sup> Münch. Med. Woch., Jan., 1913.

tecting influence in the area of ulcer. To explain this, let us turn to the doctrine of Weinland, viz.: that there is a protecting anti-body developed in the stomach for the preservation of its lining. But after accepting this we find remaining the need of an explanation for the disappearance of the antibodies in isolated regions wherein round ulcer develops. This is the unknown element in the etiology of gastric ulcer.

How are we to account for the occurrence of the localized edematous infiltration and focal hemorrhagic necrosis heretofore described? Not from local infection for, as Block has shown, the diseased structure is often singularly free from pathogenic bacteria; not from trauma, as this is generally non-existent; not from thrombosis or embolism, for their presence in the stomach is rare. Would it not be more reasonable to assign the appearance of these preliminary or primary tissue changes to some neurotrophic influence, and their subsequent digestion to the lack of the normal immunizing antibody of the tissues? I am inclined to answer in the affirmative.

What part of these disturbances is to be attributed to special vagal irritation occurring in vagotonic individuals? The answer to the question is not perfectly satisfactory.

When we take into consideration all the facts hitherto rehearsed, it has seemed to me that the best explanation for this local disappearance of resistance in the stomach may be found by including in the group of factors of the pathogenesis, the element of trophic neurosis. Through the influence of the nervous system the circulation may be temporarily or continuously disturbed, thus leaving the part vulnerable to the action of the gastric juice; or, including Weinland's theory, through the nervous system

the development of antibodies in a certain region might be inhibited as the result of which the tissues would be attacked and round ulcer would follow. I am aware that this suggestion has met with opposition in certain directions, but it has never been satisfactorily excluded from the possibilities of the case. I believe that the essential factors in the cause of gastric ulcer include certain systemic predisposing conditions among which are chlorosis and hyperchlorhydria; that resistance to digestion is lost in certain small areas through the intervention of a trophic neurosis interfering with the circulation or the normal nutrition of the part. As an illustration of what is meant in this use of the term "trophic neurosis," I would suggest herpes as satisfactory.

It is in the nature of herpes to attack certain portions of the body by preference, as for instance, the lip or the prepuce. It is prone to recur repeatedly in the same vicinity. Some individuals seem predisposed to it, especially when the health is deteriorated. It is obscurely related to the infections. The experiments of Turck, instead of excluding, rather confirm this origin of gastric ulcer. I would not insist on herpes as the precise precursor of gastric ulcer; on the other hand, it may result from angiospasm, or from other circulatory disturbance. It is not strange that ulceration can be produced in the gastric mucosa by a variety of causes. The mystery is that peptic ulcer should be so reluctant to heal. It belongs to a class by itself, and its history strongly points to a trophic cause.

Peptic ulcer then includes in its cause and its nature some other factor and quality than a local injury, an infection or an excessively acid gastric juice. Though we may not exclude these as elements in the cause, they are but steps in a series, of which the chief is some influence that removes from the mucosa its inherent resistance, something that, when present, enables it to endure a hyperchlorhydria and when absent, leaves the mucous membrane and the other gastric layers an easy prey to the digesting, eroding gastric juice even when the secretion has but feeble activity.

At present there is a gap in the etiology of gastric ulcer, and we have only an hypothesis to bridge it.

## SUMMARY

In reviewing all the evidence bearing on the pathogenesis of peptic ulcer, certain general principles stand out convincingly.

- (1) There must occur a localized devitalization of some area in the gastric mucous membrane.
- (2) With devitalization and an active gastric secretion there occurs a digestive destruction of the unresisting tissue.
- (3) There exists in the normal stomach an inherent immunity against autodigestion.
- (4) Any one of several pathological processes may be adequate to give rise to localized devitalization of tissue, be it partial or complete, transient or continuous.
- (5) Both the structure and function of the pylorus predispose it to peptic ulcer. This refers not alone to the pylorus, but to the prepyloric area and to the upper inch or two of the duodenum. As we have seen, the structure of these parts is intricate and highly specialized.
- (6) The evidence indicates that any one of various agencies may bring about localized predisposing structural defects. But after marshaling all the better understood causes that explain these primary local injuries to structure there yet remains to be accounted for the larger

number of cases, those of the familiar type of peptic ulcer. To explain these it seems necessary to include as cause either some derangement of the internal secretions that leads to cytolysis in the mucosa of the region especially attacked and comparable to the changes produced experimentally and hitherto considered; or else a trophoneurotic disturbance analogous to herpes.

There is no pathologic inconsistency in assuming that these two are jointly responsible; that the nervous disturbance leads to trophic changes and that these, in turn, interrupt the formation of antipepsin, or contribute to the formation of cytotoxic substances. While realizing the insecurity of opinion based on what for the time being remains hypothetical, I am strongly impressed with the probability of a neuropathic background for peptic ulcer.

## CLINICAL COURSE AND SYMPTOMATOLOGY

Case XI.—The patient was a housemaid of 19, having a remarkably beautiful skin, with high color of cheeks and lips. Though of a nervous temperament she had enjoyed exceptionally good health. However, she became chlorotic and constipated and lost appetite. She was seized with gastralgia strictly localized just below the ensiform radiating thence to the back near the left border of the spine. The pain occurred soon after eating, gradually subsiding to return after the next meal. There was no stasis. There was tenderness to pressure in the epigastrium. She vomited slightly cloudy, very acid fluid. A few days after the onset of pain she fainted and vomited a large quantity of bright blood.

The case was diagnosed as acute peptic ulcer, located at the lesser curvature and the posterior wall.

Morphin and atropin were used hypodermically, and

the stomach was put at rest for a few days; then thin gruels were given and subsequently a milk diet until all symptoms had disappeared; meantime the patient was kept continuously in bed. The cure was complete, the chlorosis disappeared, and the patient had no recurrence of the ulcer.

Case XII.—A married woman of 28, was the mother of three children. Previous to her marriage at 22 she had had no stomach trouble. The symptoms of ulcer began with the cessation of lactation with the first child, and continued until she again became pregnant. This relief from symptoms during pregnancy and lactation was three times repeated. When she ceased nursing her children she began to suffer from an irritable stomach, waterbrash, irregular appetite, nausea, vomiting, stasis, intense pain and local tenderness to pressure at a point just to the right of the midline of the epigastrium. pain became almost unbearable about two hours after eating. There was a brief respite after vomiting, but suffering was more or less continuous, although not directly excited by eating. With each attack at length there occurred profuse and repeated gastrorrhagia.

The case was diagnosed as acute gastric ulcer near the pylorus.

In spite of the perilous symptoms, the hemorrhage and racking pain, it was found impossible to subject the patient to a proper state of rest. After a few days starvation in bed, she went about her household duties. Great relief was afforded her by an antacid mixture. She gradually recovered, and remained perfectly well until the ulcer recurred two years later. From the second attack she recovered, and a year later from a third, each attack immediately following the cessation of lactation. With each attack she had hemorrhage, and the same un-

usually severe pain. I regard this case not as chronic ulcer, but as one of recurring acute ulcer.

Case XIII.—A middle-aged man, at the time of and immediately after eating, suffered pain at the lower end of the sternum and between the shoulders. Vomiting or regurgitation usually followed. There was difficulty in swallowing and a sensation of the food sticking, apparently the result of spasm at the cardia. On one occasion following severe retching there occurred extensive hematemesis.

The case was diagnosed as ulcer at the cardiac end of the stomach.

The patient was required to rest three weeks in bed and to take a strict milk diet; there was recovery, except that the cardia remained unusually susceptible to irritation.

In rehearing these three cases my object has been to describe definite types of acute ulcer affecting respectively the lesser curvature, the pylorus and the cardia. This list, of course, does not include all types of recent ulcer, but it serves to present instances for reference. Since ulcer expresses itself very differently in different cases, the diagnosis as to location is not always to be made without reservation.

The first case cited represents the common type of recent ulcer. Appearing in this way the symptoms are classical. The symptom-complex is essentially that of painful dyspepsia, acid vomiting and localized burning pain and tenderness. To this may be added hemorrhage so gross that its occurrence can not be overlooked. In the majority of cases, there is hyperchlorhydria, although stasis and attacks of pyloric spasm are less frequent than with ulcer at the pylorus. Before the occurrence of pain, the appetite is usually good, although with the develop-

ment of pain, the patient sometimes becomes reluctant to eat from fear of inducing suffering. There is sometimes nausea and vomiting immediately after taking food, a symptom which may be marked even when the pain is only moderate. There is great difference in cases as to tolerance of food. It is noticeable that there is no repugnance to meat, a matter worthy of consideration because this is contrary to the usual behavior in early cases of carcinoma of the stomach. The patient with gastric ulcer does not show cachexia and is rarely much emaciated.

The Onset of Recent Ulcer.—At times, preceding the more definite symptoms of ulcer, there is the history of dyspepsia for weeks or even months. This early stage is characterized by distress soon after eating with eructations of gas and water-brash. Following this, upon examination after a test-meal, the stomach may show delay in emptying. Nevertheless, the appetite is often good and the patient is well nourished. In a proportion of cases there is also definite pain two or three hours after This is apparently associated with the heightened spasticity of the pylorus, as the acid chyme passes into the duodenum. In other instances, as in the first case, the patient is practically without symptoms before the onset of the syndrome which spells ulcer. In a still smaller proportion of cases, the first intimation of the disease is the occurrence of faintness or even syncope, associated with the vomiting of blood and the passage of tarry stools.

Many times it is impossible to decide exactly when the ulcer began, although painstaking questioning of the patient may show that there has been an irritative dyspepsia of some duration. By avoidance of leading questions and with sufficient patience, a fairly accurate idea may

be formed of the date when the ulcer made its appearance.

Irritative Dyspepsia.—The question is often asked. how can the irritative symptoms that act as fore-runners of peptic ulcer be distinguished from those of a purely functional hyperchlorhydria? A reply to this is found when we remember that hyperchlorhydria is for the most part a condition depending upon some definite cause. We should, therefore, attempt to eliminate other sources of the disturbance, thus reaching a diagnosis by exclusion. For instance, if the gastric symptoms appear in connection with constipation and torpidity of the liver, and disappear promptly when those are corrected, the origin of the trouble is probably not in the stomach but in the cecum. If the symptoms are associated with those of irritation of the peritoneum and intestine and auto-intoxication, the cause is probably a chronic appendicitis. accompanied by languor, feverishness and a sensation of tension about the right hypochondrium, the hyperchlorhydria may be attributed to the biliary apparatus. is an irritating eye-strain accompanied by anxiety. insomnia, or other striking functional disturbances, the stomach trouble may find explanation in ocular defect.

The irritative dyspepsia preceding gastric ulcer usually has a more definite beginning than other types of dyspepsia. It is made worse by slight dietetic indiscretion; it frequently gives evidence of over-sensitiveness of the pylorus, as may be recognized from the fact that the distress appears some hours after eating, for instance, in the middle of the night. It must be said, however, that it is at times almost impossible to differentiate between the dyspepsia occasioned by ulcer and that coming from other causes. This is especially true when the ulcer develops in a patient who has already suffered from an irritable

PAIN 275

stomach, the result of a highly impressionable nervous system, or gastric hyperesthesia. Sooner or later gastralgia develops in the majority of ulcer cases, and this usually marks the beginning of erosion of the gastric mucosa and over-tonus of the muscular layers.

Pain.—About the earliest definite symptom of open gastric ulcer is the occurrence of pain lasting for an hour or two after the ingestion of a hurried meal, or after the taking of food that demands unusual activity of the gastric digestion. The hurried luncheon of the ordinary American business man is an illustration of what I mean. Pain may be excited by drinking spirits or by taking highly seasoned food that stimulates gastric secretion and motion. More frequently it follows the eating of pastry, strawberries or foods that are rich in fatty acids. frequent distress experienced after eating fried food'is due to the formation of fatty acids. Idiosyncrasy seems to play a part in these cases in reference to diet. It is characteristic of the pain of ulcer to be definitely localized, and there is usually sensitiveness to pressure over a small area which corresponds with the greatest intensity of the pain. Generally this is located precisely in the pit of the stomach, sometimes a little higher up, just under the ensiform, depending somewhat upon the conformation of the patient. It apparently does not depend upon the precise location of the ulcer. As a rule a second point of tenderness can be made out a little below the pit of the stomach and to the left of the median line and may depend upon a heightened sensitiveness of the solar plexus. Along the border of the aorta, in case there is a thin and sufficiently relaxed abdominal wall, tenderness may be elicited from point to point by steady, deep pressure, doubtless from irritability of the abdominal sympathetics. Identical results are found in those who are hysterical and hyperesthetic, and the sign is of little importance unless one takes into consideration the general characteristics of the patient. The pain of ulcer is usually described as burning or boring in character. I have sometimes thought that this description of the pain depends in certain cases upon a preconception in the mind of the physician. When interrogated with sufficient care, the patients describe the character of the pain in very different terms. Here again there is danger of error for the reason that many persons select their words indifferently and without discrimination as is exemplified in their halting efforts to convey to another a precise description of any unnatural sensation. The pain in ulcer often takes an anteroposterior direction, extending from the pit of the stomach toward the spine. Sometimes there is a definite backache just at the left of the spinal column, and in nearly every case, upon careful examination, there can be determined in that location a definite area of ten-Most often this is located in the eleventh left dorsal interspace, about an inch from the spinal process; sometimes as far up as the ninth dorsal, sometimes as low as the first or second lumbar interspace, but especially on the left side. Occasionally the pain radiates very widely, but usually upwards rather than downwards and to the left rather than to the right. I have met with several cases in which there was sternal anguish that suggested angina pectoris or aneurysm. The pain is neuralgia, dependent on irritation produced by the acid gastric juice attacking exposed nerve filaments and upon gastric over-tonicity. It suggests toothache which follows dental necrosis and, like toothache, it may be excited by the taking of hot or cold fluid, especially cold, and may appear suddenly and as suddenly disappear. The pain of gastric ulcer is not relieved by pressure; on the conPAIN 277

trary this usually makes it worse, so that the wearing of a tight belt or of corsets increases the suffering. Women often say: "The pain was so severe that I had to take off my corsets." Like all neuralgias having a fixed cause, the pain is exaggerated by anxiety and other worrying psychic states. It is sometimes aggravated just preceding menstruation; it is sometimes excited by exercise. A man having ulcer said to me: "The pain comes on when I am walking, but lets up when I sit down." The pain of gastric ulcer seems to be associated with a state of over-tonicity of the stomach walls which explains the fact that relief may be obtained from the use of hot fomentations. In addition to over-tonicity, there develops after a time an over-contractility, and this in turn, may develop into gastrospasm. This spasmodic contraction may be confined to a part of the stomach, affecting only muscle fibers of a certain zone, or it may be widespread and accompanied by sympathetic feelings of constriction or suffocation over the precordium or portions of the thorax. Sometimes there results irritability of the bladder and excessive urination. The element of spasm is displayed more especially in the region of the pylorus. There the muscle walls are thickest, the innervation is most complex, the gastric acidity is highest and there the ulcer is most likely to be located. This is illustrated in Case XII. Sometimes the pain is of minor importance, except when the spasm occurs; and this is apt to be in the night, or two or three hours after eating. Though attributable in part to the heightened acidity natural at such times, it depends mostly upon spasm. course hyperacidity or over-secretion are involved because the more the pylorus is stimulated the more spasticity is excited, and it is agreed that the antrum of the pylorus is for the most part excited by excess either in acidity or in quantity of its contents. This explains why both spasm and subsequent pain are relieved by the taking of alkalies.

It is commonly stated that delayed pain is characteristic of duodenal ulcer, but it is likewise usual in pyloric and prepyloric ulcer, although perhaps to a less striking degree.

Over-acidity and Over-secretion.—Estimation of gastric acidity should be made from contents withdrawn by means of the stomach tube. The making of a chemical examination of the vomitus does not answer the purpose. The fact is well known that the physical and chemical characteristics of vomited matter differ considerably from those of contents aspirated after a definite test meal. Some misapprehension has arisen from the fact that vomited matter has been used for the study of gastric chemistry.

Formerly there was much reluctance to use the stomach tube when an open ulcer was suspected. I have convinced myself that such hesitation is usually groundless. Of course the reservation should be made that only the proper kind of stomach tube should be used and that in the hands of a skilled person. The instrument should have a blunt end; should be fenestrated near its lower extremity; should not be too rigid and should be of large caliber, not under 35 French for an adult. The careful use of the stomach tube is in most cases not only without danger, but is often of great value in the study of gastric ulcer. When there is no obstruction at the pylorus and when there is not too prolonged spasm, the digestion proceeds without delay and the animal proteid disappears somewhat more quickly than in health. When there is stenosis at the pylorus or pylorospasm, there is delay in the emptying of the stomach with resulting stasis of the gastric contents. In these cases particularly we find hyperacidity and often too much fluid pours from the tube. Whereas in health, one hour after an Ewald-Boas test meal, only 50 to 90 c.c. of liquid or semi-liquid contents are recovered, in ulcer 100 or 200 c.c., or even more, may be withdrawn. In relation to this excess in amount of contents much discussion has arisen as to whether the increase is due to stasis or to over-secretion. While some have regarded it as the result of gastrosuccorrhea, others look upon it merely as retention. Soupault, who operated upon 28 cases of so-called gastrosuccorrhea, found ulcer located in the pyloric region in all. From such reports has grown up the belief that gastrosuccorrhea is merely an expression of stasis from pyloric obstruction. Though there is some ground for this conclusion, it is undeniable that gastrosuccorrhea may also occur when there is neither ulcer nor obstruction at the pylorus. Sufferers from gastrosuccorrhea, on account of an excess of acid gastric juice, are admittedly exposed to one of the contributing causal factors in the development of ulcer. In actual practice, when an excess of gastric juice is withdrawn after a definite meal, we conclude that there is spasm or stenosis at the pylorus and this decision is usually correct.

There are cases in which the stomach empties itself with unusual celerity. Riegel was quite clear on that point, and I have repeatedly verified the fact. It was formerly believed that hyperchlorhydria was almost uniformly present in recent gastric ulcer, but of late years we have come to modify this belief, perhaps as the result of more frequently repeated use of the stomach tube in these cases. At any rate, we no longer regard hyperchlorhydria as a necessary, although it is a usual concomitant condition. Ewald states that he finds great

variation in the total acidity from time to time. I have observed the same in my own cases. In a group of 99 cases Ewald found hyperacidity in 34, normal acidity in 56, and hypoacidity in 9 cases. My records on this matter place the hyperacidity in somewhat higher proportion, although I hesitate to state this in precise figures. Although some of my cases have had numerous analyses, others have undergone but one or two. That is the source of difficulty and inexactness in attempting to decide such questions by statistics. We need to know not alone the exact number of cases, but the nature and number of test meals, the stage of the ulcer, the relationship in time to preceding periods of fasting, dietetic abuses of the stomach and the occurrence of hemorrhage. As nearly as I can conclude, after a review of my cases, at least 50 per cent of all show hyperchlorhydria and not more than 5 per cent show subacidity. Riegel believed that hyperchlorhydria is an important feature in the evolution of typical gastric ulcer. After all, that is the main point of the discussion and in his position Riegel was right. Lactic acid very rarely is found present, except when carcinomatous degeneration has become well established in the domain of a preceding ulcer. The increase of lactic acid under these conditions is sometimes very significant. I had occasion to study this question accurately in the case of a distinguished physician. He suffered from hyperchlorhydria and moderate stasis, having at times intense pylorospasm. Under treatment his symptoms abated, the free hydrochloric acid dropped gradually from a point above 100 to 50, whereupon the patient felt comfortable. Then lactic acid appeared, at first only in traces; the hydrochloric acid continued to fall, but in spite of this there was a renewal of the symptoms of irritation and pyloric spasm. Meantime the patient lost rapidly in weight, showed cachexia, and the diagnosis of carcinoma developing upon ulcer was made. The patient succumbed in a short time without operative interference. At autopsy there was found a typical round ulcer near, and somewhat involving, the pylorus. A small area showed ingrowing cancer.



FIG. 20.—Specimen Showing the Pylorus from Case Described. The canal may be seen to be almost closed by cicatrix of ulcer, the ingrowing carcinoma, showing in the center and at the lower border of the picture, may be recognized by its lighter shade.

It was surprising to discover how small an infiltration of carcinoma could bring about such a marked change in gastric chemistry. This presence of lactic acid is, nevertheless, an unsatisfactory criterion of the development of cancer. Sometimes it is absent and occasionally, although not often, in peptic ulcer, lactic acid is found when cancer is not present, a statement that should not be too much emphasized.

Gastritis.—Although there is a local inflammatory reaction at the border of a peptic ulcer, evidence of gastritis is not frequently met with in the stomach contents except in case of the alcoholic. The finding of mucus in flakes or in little masses intermixed with the stomach

contents together with an excess of degenerated epithelial cells and leucocytes, points toward the presence of gastritis.

Hemorrhage.—The occurrence of hemorrhage, hematemesis and melena, is one of a triad formerly so much paraded in the history of ulcer. Undoubtedly considerable hemorrhage can occur without hematemesis, while melena passes unobserved. On the other hand, hemorrhage is reported mistakenly. One night I was hastily summoned to see the wife of a physician, being told that she had gastric ulcer with hemorrhage. I found a much frightened young lady complaining of pain in the stomach. Then I was shown a receptacle containing a dark stained material which, upon examination, proved to be vegetable matter. It was ascertained that the patient had recently eaten heartily of purple plums. She has shown no evidences of ulcer since that time.

Hemorrhage in ulcer cases is reported by several different observers to occur in the following proportion: Brunton, 29 per cent; White, 30 per cent; Gerhardt, 47 per cent; Müller, 29 per cent; Lebert, 21 per cent; von Leube, 46 per cent; Ewald, 54 per cent; Joslyn, 81 per cent.

Again I am reluctant to state my own figures. Of course it is understood that the above tabulation refers to gross hemorrhage in which either hematemesis or melena or both have taken place. It does not refer to small hemorrhages discoverable only by means of tests for occult blood. I believe that gross hemorrhage occurs in about 33 per cent of all cases. But when we consider how frequently gastric ulcer passes undiagnosed, and how often hematemesis occurs, which depends upon some cause other than gastric ulcer, although mistakenly attributed thereto, it will be seen that exact statistics are

open to suspicion. Hemorrhage is not properly such an alarming manifestation as it is commonly reputed to be. Until recently it gave me very little concern, for under personal care I had never lost a case of gastric ulcer from this cause. Lately, however, I have had two fatal cases. There is a difference of opinion as to the frequency of death from this cause. Ewald states that out of 360 cases of his own, 1.2 per cent died from hemorrhage. He quotes Rodman as having 8 per cent and Bramwell the same.

The fatalities from hemorrhage occur for the most part in aged persons, whether because of higher blood pressure and vascular degeneration I do not know. It is partly owing to the fact that chronic ulcer with deep-seated erosion, with extensions into surrounding parts, and with opening into larger vessels is met with much more often in the old man than in the young.

Melena.—It is advisable to instruct patients who are suffering from irritative stomach symptoms to make intelligent observations of the stools. Undoubtedly melena is not always recognized because of the repugnance which many people have for scrutinizing the stools and the lack of understanding on their part as to what is important to note and report upon. Patients sometimes fall into error through regarding as melena the sanguinolent stools that result from bleeding hemorrhoids. In the female the source of the blood may be mistaken to be in the intestine when, in fact, it comes from the uterus. It alarms the patient less, and is less likely to lead into error, if we avoid the use of the term "blood in the stools." It is better to make the patient understand the appearance of blood-containing evacuations as changed through digestion in the upper intestine; in other words, melena. Having done this we must teach him to discriminate between

melena and the stools discolored by drugs or by coloring matter in the food. It may seem needless to emphasize the importance of the inspection of the stools by the physician. Yet all will admit that this is frequently neglected, and through this oversight we are often led astray. The impression still survives that melena is characteristic of duodenal hemorrhage and the vomiting of blood of gastrorrhagia. Of course melena is characteristic of gastric as of duodenal hemorrhage; and for that matter may be a sign of hemorrhage arising in still other regions: for instance, the esophagus, the nasopharynx or the bronchial tract. Melena may and often does result from gastric ulcer, without being associated with hematemesis. and on the other hand, one may see the abundant vomiting of blood in cases of duodenal ulcer when there is found comparatively little evidence of it in the stools. Practically, however, we always find melena when there is hemorrhage of importance, coming either from the stomach or the upper intestinal tract.

Occult Blood.—The microscopic examination of the dejecta is of little practical use when the hemorrhage comes from the upper digestive tract, but the chemical search for hematin in the stools is of great importance. An open ulcer in the stomach or the duodenum leaks a little blood at least occasionally. In cases of suspected ulcer a daily search should be made. One or two examinations are not as valuable as a series carried on every day for a week or longer. This test is less reliable when applied to stomach contents. The presence or absence of occult blood is not a criterion even in the stools, for although it contributes valuable evidence it is open to exception. The subject is well discussed by H. W. Soper. 18

Vomiting.—Although vomiting is a feature of gastric

<sup>18</sup> Interstate Med. Jour., March, 1912.

ulcer, it is, like hematemesis, by no means an invariable symptom. At least occasional vomiting occurs in about one-half the cases, and nausea is present in a still larger proportion. In a small group vomiting is the most persistent and striking symptom. There may be anorexia, but sometimes the patients have hunger, although the food is vomited as soon as swallowed. Gastric ulcer with vomiting as the persistent symptom, a type of the disease to which Lebert drew attention, although uncommon, should be recognized, as the vomiting is not infrequently attributed to other causes. In classical gastric ulcer vomiting is, however, occasional and not persistent. vomiting is usually associated with pain and occurs when the pain is at its height. Commonly there is a mitigation or cessation of pain when the stomach has emptied itself. The frequency of vomiting and the amount ejected usually depends upon quality and quantity of food Distension of the stomach from any cause may be sufficient to produce vomiting, or it may follow the taking of solid or stimulating liquid food, and yet not result from the ingestion of bland fluids. At times emesis takes place soon after meals, giving rise to the characteristic "food vomiting." When the ulcer is in the pyloric region, pain and vomiting are apt to occur some hours after meals, and the ejecta may be food-free or contain only particles of starchy or vegetable matter. fibers and proteids generally show the effect of active digestion and may be in perfect solution. The vomitus is sometimes grass green in color, sometimes yellowish, sometimes merely a thin watery mucus, grayish or without color. When the retching is prolonged one often finds material coming from the duodenum as shown by the presence of bile. Generally the patient volunteers the statement that the ejecta are sour to the taste; sometimes they are intensely sour. I have noted this complaint even when the material subjected to titration was found to be of rather low acidity. At times there is met with a case in which vomiting occurs in the night or early in the morning. This may depend upon food stagnation, as is shown by the abundance of the vomitus and by the food which it contains, or upon gastrosuccorrhea. In the latter event the emesis is not only apt to occur early in the morning but the material discharged is relatively foodfree. One finds a quantity of pale, gray, thin, watery mucus having high acidity and separating upon standing into two layers, the upper being comparatively clear, and the lower having a pale granular appearance.

After all too much importance must not be attributed to vomiting in gastric ulcer. It does not occur in more than 50 per cent of even advanced cases. Besides this, vomiting is common to so many diseases aside from ulcer that it is unwise to insist too much upon its importance as a symptom.

## ULCER WITH ABRUPT ONSET

Sudden hemorrhage, though sometimes occurring alone, is commonly but one of a group of symptoms in cases that have an abrupt onset. Here there is an absence of the preliminary train of symptoms that are characteristic of typical gastric ulcer. Without premonition, the patient is usually seized with pain, localized tenderness and vomiting of blood. The subsequent history of these ulcers may be that of early recovery or, notwithstanding an abrupt beginning, they may be obstinate and become chronic.

## RECURRING ULCER

In a certain group of cases there is an acute beginning followed by the typical symptoms of ulcer, and apparently a complete recovery after a few weeks. The patient remains symptom-free for months or even years, during which time the stomach demands no especial care. Then the ulcer recurs in precisely the same manner; again recovery takes place, and after a time there is another recurrence, as in Case XII. Sometimes two or three years elapse between the attacks, and these repetitions may go on for a lifetime. The following case is an excellent illustration:

Case No. XIV. Hyperchlorhydria, Pain, Gastrosuccorrhea Periodica, Moderate Hemorrhage, -An Englishman. aged 37 years, unable to eat meat until he was 28 years old; then forced himself to overcome the repugnance and without noticeable effect upon his health. 18 years he has had recurring attacks of peptic ulcer. never going longer than two years without an attack. His present illness began three weeks ago, with epigastric pain, especially on waking in the morning, sometimes accompanied by regurgitation of sour fluid, eructation of gas, local epigastric tenderness and occasionally the presence of melena. The pain occurs from one to two hours after meals and is usually relieved by taking food. total acidity varies from 100 to 140, depending mostly upon free HCl. The patient has always recovered by following a prescribed diet and the taking of antacids. He has never discontinued his usual vocation, that of chemist.

#### CHRONIC ULCER

The clinical history of chronic peptic ulcer in some respects so closely corresponds to that of the acute or recent type that a detailed description here would cause unnecessary repetition. I believe that most chronic ulcers begin as acute ulcers, and therefore, for a while, it is proper to regard them as such. It is only later that, as the result of cicatrization, the aggregation of inflamma-

tory tissue, the increased rigidity of the prepyloric and pyloric regions, perhaps the advent of perigastritis, that symptoms of a chronic nature appear. When the stomach is anchored to surrounding parts, when the ulcer perforates or, burrowing, invades other organs, when the intervention of infection leads to the formation of subphrenic abscess, the symptomatology of chronic ulcer is fully displayed. Undoubtedly in part, these symptoms relate to the results of ulcer rather than to chronic ulcer itself. Frequently the attempt to relate the symptoms to the various stages of the process is unprofitable.

The following case is typical of chronic ulcer ending in cancer.

Case No. XV. Chronic Peptic Ulcer at Pylorus, Stenosis, Terminating in Carcinoma.—The patient, 50 years old, had led an exemplary life and had never been ill previous to the past two years, when he began to have an irritative dyspepsia. There was sour stomach, gastric distress, a sense of weight in the epigastrium and eructations of gas. He found little relief by treatment or diet. Although he consulted several physicians, the correct diagnosis apparently was not made. He rarely vomited; there was no hemorrhage; the epigastric distress was occasionally varied by severe pain. At length this became unbearable, whereupon he presented himself at the clinic for treatment. At this time the pain, especially severe at night, radiated to the back, where there was tenderness at the tenth interspace near the spine on the left side. Occult blood was found constantly in the stomach contents and in the stools. There were neither lactic acid nor Oppler-Boas bacilli; free HCl was present in excess. The patient was relieved by an appropriate diet, antacids and lavage. A cure seemed probable, when somewhat unexpectedly, he began to show evidences of food

stagnation with renewal of symptoms. By very careful palpation, a small mass could be recognized at the border of the right rectus muscle. The patient was moved to the Buffalo General Hospital where, on exploration, there was found an enormous mass of connective tissue in the prepyloric and pyloric regions extending to the duodenum, binding the parts down with scar. The appearance of the mass, and the finding of two large lymph nodes, led to the belief that carcinoma had invaded the ulcer, and gastro-enterostomy was performed; the patient died two weeks later. At autopsy the mass seemed to be composed of old scar and an accumulation of recent inflammatory tissue. After prolonged histological study there was found a small, definitely outlined, carcinomatous infiltration in the submucosa at the border of the ulcer. No fresh erosions were discovered. The exacerbation of symptoms was therefore not dependent on renewed activity of the ulcer, but on the beginning of cancer. slow beginning, as in this case, the persistency of symptoms, the obstinacy to medical treatment, and the deformity occasioned by inflammation and adhesions, producing motor insufficiency, are familiar passages in the history of chronic gastric ulcer. The change for the worse after a period of improvement is typical of ingrowing cancer. Chronic ulcer, like acute ulcer, when located at the cardia, excites pain and spasm upon the ingestion of any but the blandest food, as in Case XIII, so that emaciation from starvation is often extreme.

The appearance of spasm at the cardia is not always a positive indication that the ulcer is at or near that point. In two cases recently under my observation in which cardiospasm developed with signs of ulcer and in which gastrostomy was performed for the purpose of nourishing the patient, it was discovered that there was also ob-

struction with ulcer at the pylorus. A gastro-enterostomy was necessary in order to relieve the patient. After gastro-enterostomy, and not before, the cardio-spasm disappeared. When an ulcer is situated between the fundus and the antrum pylori there sometimes develops, as the result of cicatrization, the so-called hourglass or bilocular stomach. (See Radiogram VIII.)

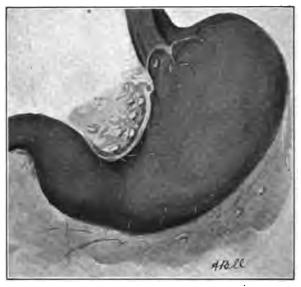
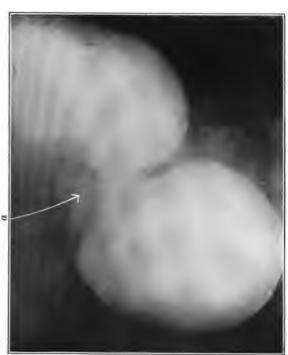
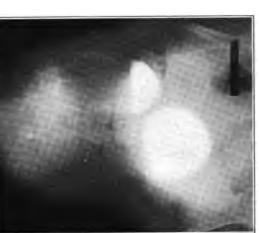


FIG. 21.-HOUR-GLASS STOMACH.

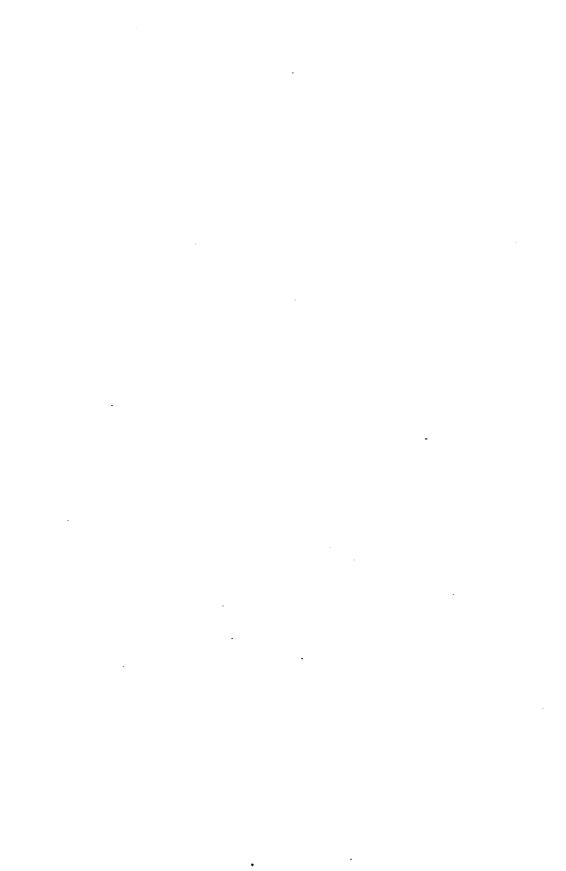
Hour-Glass Stomach.—This deformity may be static, because of the density of the scar or because of adhesions to the liver and other parts, and then great narrowing of the stomach at this isthmus may follow. The ulcer may heal, leaving the patient with serious embarrassment to digestion owing to interruption of gastric peristalsis. Great care has to be exercised in eating.

There is irregularity of motor function and consequent eructations of gas and sour regurgitations. Sometimes there is emesis and the patient is surprised to find





was engaged by strong perigastric adhesions and drawn by these into a mass of sear tissue at the lesser curvature. There existed incomplete obstruction both at the isthmus, or the seat of the ulcer, and at the pylorus, as the result of perigastric adhesion and traction. Hence both pouches were distended. Relief was obtained by anastomosing the two pouches and by making a posterior gastro-jejunostomy with the second pouch. Subsequently a diaphragmatic pleurisy developed as a result of infection through the Radiograms No. XIII and XIV.—Hour-glass Stomach. Radiogram XIII, standing posture. Radiogram XIV, lying posture. The small shadow (a) is produced by the escape of bismuth through a slow perforation which occurred at the lesser curvature, presenting the appearance of a small diverticulum. When the patient was operated upon it was found that the pyloric end of the stomach Radiogram No.  ${
m XIV}$ Radiogram No. XIII. diverticulum.



a striking variation in the character of the vomitus. At first there is brought up food but slightly digested, but after retching the vomitus becomes fluid, and intensely sour or bitter. Several times I have had the experience of washing out the stomach until it appeared to be clean and thereafter the patient would vomit material quite unlike that which had just been recovered. One time the stomach contents were of low acidity but upon manipulating the tube and pressing it down farther it evidently passed into the second compartment of the stomach, for there was withdrawn contents of an entirely different color, showing advanced digestion and, upon titration, very high acidity. Occasionally the biloculated stomach performs its function without great inconvenience to the patient, but usually the persistency of symptoms makes advisable a wide anastomosis between the two pouches. In a proportion of cases the biloculation is not a persistent, but transient condition. This results from the fact that an open ulcer causes irritation and consequent spasm in a narrow zone, thus dividing the stomach temporarily into two parts. In such patients, with healing of the ulcer, complete recovery is possible.

Pyloric Stenosis.—One of the most frequent ill-results of chronic ulcer at the pylorus is stenosis with subsequent food stagnation and gastrectasis. Corresponding symptoms without stenosis may result from spasm in recent ulcer, but the spasm subsides after proper treatment, and the symptoms disappear. Of course it is conceivable that when an acute ulcer has passed and gone there may remain sufficient cicatrization to obstruct the pylorus permanently. However, this conclusion should not be reached too quickly. Repeatedly I have examined patients of whom it had been said that the pylorus was so constricted that nothing but operation could relieve

it, and have found that under proper and sufficiently continued medical treatment all symptoms of obstruction and motor insufficiency disappeared, leaving the patient apparently well. In recurring ulcer at the pylorus there is often temporary food stagnation. I have now under observation a patient who first came to me suffering in this way twelve years ago. In his case there was not only food stagnation but moderate dilatation of the stomach. His severe gastric distress continued until relieved by vomiting of large quantities, after which he was comfortable for twelve or eighteen hours. quickly relieved by a diet of milk and gruel, the administration of antacids and by daily lavage. He remained well for a year in spite of the fact that he was indiscreet in the manner of living. Then the unpleasant experience recurred, together with evidences of open ulcer. At four distinct periods he has suffered in this way and has as often recovered. I formerly advised operation, but he declined it; now I advise against it. In connection with stenosis or with other benign obstruction the question of gastrosuccorrhea or Reichmann's disease often presents (See Page 481.) itself.

Perigastric Adhesions.—Perigastric adhesions are usually of less importance than is commonly believed. It is difficult to decide to what extent symptoms are thus occasioned. When there is persistence of eructation and other motor symptoms, or of irregular pain, it is natural to accuse perigastric adhesions of being the exciting cause. This is notably true of those cases which have undergone operation and which have not received therefrom the complete relief which was expected. The surgeon in his perplexity, satisfied with his technique, finds in supposed adhesions an explanation for the continued trouble. Undoubtedly he is sometimes right in this conclusion, but

as often he is mistaken. The irritation may depend upon the continuance of the ulcer or upon some undue tension from scar in the walls of the stomach. In not a few cases, the symptoms are but the expression of a neuropathic, or psychopathic, condition which has become exaggerated through the sensational events connected with a major operation. It is well-known that the tissues of the peritoneum are prone, in certain individuals, to form adhesions, after even slight manipulation. I have met with several exaggerated cases in which operation after operation undertaken with the object of breaking up adhesions, has been followed by no relief or at best only by temporary improvement. In fact, as a rule, these patients are made worse by each succeeding operation.

Sometimes the stomach is adherent over its entire surface. I have known this to be the case with the development of no other symptoms than a feeling of epigastric fullness after a small repast. It is said that when the greater curvature is bound down the lower border of the stomach is not depressed as much as usual upon insufflation of the stomach. Perigastric adhesions at the pylorus or just beyond may lead to sharp bending of the duodenum or, by contracting the canal, make it so narrow that a degree of obstruction occurs. Adhesions which develop around the lesser curvature, binding the stomach to the pancreas posteriorly or to the left lobe of the liver anteriorly, may occasion severe pains which radiate towards the back, the left chest or shoulder. This suffering is usually increased when the patient assumes the upright position and is relieved when he lies down. In such patients one may find hyperesthesia in the adjacent left intercostal nerves. When adhesions are very thick and are located towards the lower end of the stomach, they may sometimes be felt on palpation. Ewald looks upon perigastric adhesions as constituting one of the most troublesome chapters in gastric pathology.

#### DUODENAL ULCER

The nature of duodenal ulcer corresponds so closely to that of gastric ulcer that the subject requires but brief special consideration here.

In the Newborn.—Duodenal, more often than gastric, ulcer occurs in the very young. Melena neonatorum may occasion death and the autopsy reveal that the bleeding came from unhealed duodenal ulcer. Peptic duodenal or gastric ulcer may arise soon after birth. In these cases of melena neonatorum the ulcer is found above the bile papilla and usually below the pylorus, rarely within the stomach. As most reported cases have ended quickly and fatally, the structural changes observed are those of very recent ulcer.

Vomiting in Infants.—At times, following hematemesis in infancy, there occurs persistent vomiting accompanied by wasting. These symptoms result from cicatrization or from spasm excited by unhealed ulcer. One naturally speculates upon the possible relationship between peptic ulcer and the so-called idiopathic, or congenital pyloric stenosis of infancy. Without question some cases of incoercible vomiting of infancy depend upon ulcer, cicatrization and obstruction. In most cases undergoing operation or seen at autopsy, evidences of ulcer are wanting.

A case related by M. Torday 19 simulated in its clinical development congenital stenosis of the pylorus. There were present the signs of inanition; vomiting occurred after each nursing and was not checked by lavage or other measures. No blood was discovered in the vomitus or feces. The lower border of the stomach was two

<sup>19</sup> Bull. Méd., March 13, 1907.

fingers' breadth below the umbilicus. Peristaltic movements of the stomach were visible, but the pylorus could not be palpated. Death followed bronchopneumonia. At autopsy a typical round ulcer in the duodenum, 5 cm. from the pylorus was found. Here the symptoms of pyloric stenosis were induced by reflex spasm of the pyloric ring, secondary to duodenal ulcer.

It is hard to dislodge the suspicion that cases of great irritability of the pylorus accompanied by hypertrophy are in some way allied to peptic ulcer.

In cyclic vomiting, the possibility of peptic ulcer has often been discussed. The high acidity of the gastric secretion in these cases has favored an affirmative view; however, the autopsy evidence rarely supports it.

Obscure cases of pyloric obstruction, transient or persistent, some with and some without definite structural cause, emphasize the fact that the region of the pylorus is peculiarly conditioned; it is wonderfully endowed with functional activity, highly specialized and liable to derangement. Where there is so much complexity it is wise not to assume too much, for with further study we may have unfolded a clearer knowledge of the physiology and a more harmonious conception of the pathology of this region. More attention deserves to be given vagotonic influences. At present we do not understand why peptic ulcer in the new-born is located in the duodenum rather than in the stomach.

Following Burns.—Another peculiarity of duodenal ulcer is that it often follows severe superficial burns. Observations of this relationship reach back to the time of Dupuytren but its study has not been very fruitful. It is suggested that this sequence is not limited to any special age. Does it follow septic emboli? Just how the latter could reach the upper intestine especially is not

clear. Such embolism probably would involve the presence of a general septicemia, a condition not usually present. It has been suggested that cytotoxic substances produced by burns especially disturb the duodenum, an hypothesis which meets with less objection. Ponfick relates a case in which there were found, eighteen hours after a severe burn, hemorrhagic erosions and superficial ulceration of the mucosa of the upper part of the duodenum. This shows the rapidity with which the pathology advances; it would seem to exclude the theory of septicemia and it at least suggests the action of a cytotoxic cause.

In Uremia.—Moynihan believes that uremia is a factor in the etiology of duodenal ulcer. Uremia probably bears the same relation to this that it does to ulcer occurring in other portions of the intestine, particularly the colon. With such an ulcer arising in the first part of the duodenum, the gastric secretion unimpaired, there would probably develop a lesion having the appearance and some of the characteristics of peptic ulcer.

In the Infections.—Duodenal ulcer occurs as a complication in the infections, for instance in typhoid fever, where it appears to be specific in nature. At any rate in the cases that have come under my observation there was nothing that led to the suspicion of anything other than a typhoid ulcer occurring in an unusual part of the digestive tract. In one case in which death followed uncontrollable hemorrhage, the ulcer was located at the extreme end of the esophagus. In appearance it was identical with lesions found in the ileum in the same case. The clinical observations indicated that the ulcer began in the fourth week of the fever, a time at which gastric secretion is generally scanty and possesses little peptic activity. Typhoid ulcer might likewise attack the duo-

denum where its development would be modified by the enzymes; on the other hand, it cannot be denied that the



Fig. 22.—Duodenal Ulcer.—Large perforating ulcer of the duodenum, just opposite to which is a non-perforating ulcer.

(Museum, University of Buffalo.)

constitutional disturbances following typhoid may favor the genesis of duodenal ulcer of the ordinary type.

Location.—Peptic ulcer develops in the great majority

of cases in the upper part of the duodenum immediately below the pylorus, sometimes involving the inferior face of the pyloric ring. A compilation of 517 cases reported by several modern surgeons shows that the ulcer was located in the upper part of the duodenum in 417; in the middle portion in 37; in the last portion of the duodenum Murphy states that in 95 per cent of cases, ulcer occurs in the anterior surface of the duodenum. Although usually single, sometimes there are two ulcers at the same time, one facing the other, the so-called "kissing ulcers." At other times there is a ring of ulcers which suggests an attempt at amputation of the intestine. In a few cases ulcer develops on the posterior wall of the duodenum where the intestine is attached to the posterior abdominal wall. Perforation occurring at this point may lead to an abscess which on burrowing, may extend downward behind the peritoneal cavity, nearly to the pelvis. Such abscesses may give rise to signs of psoas irritation and the real nature of the process is readily misunderstood.

The surgeons now class with duodenal ulcer a proportion of cases which physicians have heretofore looked upon, and I think properly, as gastric ulcer. Reference is made to those lesions situated at the origin of the duodenum, or which involve the lower border of the pyloric sphincter. A sharp line is made in classification, based on the relation that exists between the site of the ulcer and the "pyloric vein." This classification seems to be too arbitrary. The question should be a clinical and functional, rather than an anatomical one. Which organ is functionally disturbed, which gives rise to the symptoms in these cases? Surely the stomach, for the manifestations are those of pyloric ulcer and the precise location it is not always possible to make out. In these cases the

symptoms are pronounced, whereas the reverse is true of ulcer located further down the duodenum.



FIG. 23—DUODENAL ULCER. LOCATED DIRECTLY BENEATH THE PYLORIC RING ON POSTERIOR WALL. Although two surgical operations were made, the ulcer was not found. Weakened by hemorrhage, the patient ultimately died from gastric spasm and a peculiar form of auto-intoxication which sometimes occurs in duodenal ulcer.

The ensuing case illustrates this; it also strikingly shows that rigid rules for determining the location of ulcer are not reliable.

Case No. XVI. Perforating Duodenal Ulcer Immediately Below Pyloric Ring; Stenosis of Pylorus; Inflammatory Thickening of Lower End of Stomach; Bismuth Retained.—Mrs. M. entered Buffalo General Hospital, July 26, 1911; married, mother of four children; had had no previous illness of importance. Wassermann, negative. For eight years had suffered from constipation, frequent and distressing distension of the abdomen, gastric distress, pain, eructations, water-brash, little vomiting. There were periods of exacerbation of symptoms; suffering had been continuously severe for 8 months, during which time she relieved herself by using the stomach tube.

A physical examination revealed a moderately enlarged stomach, other organs apparently normal. The urine was abundant; constipation, marked. Occult blood absent in stools.

Fifty minutes after an Ewald test breakfast, 200 c.c. of whitish material, quickly withdrawn: total acidity, 99; free HCl, 66; combined HCl, 23; lactic acid, 0. Occult blood, prompt reaction.

Microscope showed many starch cells, yeast cells, and a few fat droplets; occasional long and short bacilli.

Radiographic examination by Dr. Plummer: 300 c.c. bismuth mixture swallowed; exposure after 30 minutes. Stomach of "fish-hook" type, about normal size, the lower border corresponding with umbilicus; peristaltic waves marked. Pyloric extremity rounded; no bismuth had escaped into the duodenum. The shadow suggested no growth; it showed the presence of obstruction at the pylorus, the nature of which was not evident.

The symptoms were but little improved by treatment. Operation was urged, but declined. Patient deserted and took up "Christian science." Eight days later per-

foration occurred, and the patient died without treatment.

At the autopsy the stomach was found to be large, of the "fish-hook" type; the walls of the lower end of the stomach, extending 10 cm. or more, were thickened to 6 mm. The pyloric ring showed stenosis from inflammatory thickening, having an orifice the size of No. 6 French.

Immediately below the ring, located on the lateral posterior wall of the duodenum, was a chronic ulcer the size of a dime, perforating into the abdomen. There was general peritonitis.

This case has points of special interest, as follows:

The long continuance, without hemorrhage and with but little vomiting; the chronic inflammatory changes at the pyloric end of the stomach; the appearance in the radiogram being that of a stomach of normal size, although on clinical and post-mortem examinations it was found large; the absence of occult blood in the stools; the presence of hyperchlorhydria, motor insufficiency, the failure of bismuth to pass the pylorus within half an hour; constipation.

The contention that constipation is not present, that occult blood appears in the stools, that the stomach empties itself promptly, and that the Roentgen rays show a rapid discharge of bismuth from the antrum and duodenum in duodenal ulcer meets with contradiction in this case.

The accompanying cut from a photograph gives a good idea of the specimen. I am indebted to my colleague, Dr. N. G. Russell, not only for the specimen but for procuring data of the case. (See Fig. 24, page 302.)

Incidence.—By the string test Einhorn concludes that in point of frequency duodenal ulcer stands to gastric ulcer as one to four, and this does not take into consideration those cases in which the ulcer is located in the greater curvature of the stomach, in which region the string test is inoperative. Mayo Robson would make the proportion of duodenal to gastric ulcer as two to three, while W. J. Mayo considers it to be as high as three to two. If we are to judge from autopsy findings, gastric ulcer occurs



Fig. 24.—Perforating Duodenal Ulcer.

ten times as often as duodenal. This discrepancy is believed to depend upon the fact that the duodenal is more rebellious to medical treatment than the gastric ulcer, and that, as a result, surgeons do not find the cases in their normal relative proportion.

Sex.—It has long been known that men suffer more frequently than women from duodenal ulcer, but we were un-

prepared for the statement of W. J. Mayo that upon the analysis of 100 cases, 77 occurred in males and but 23 in females. He reports the occurrence of gastric ulcer as about equal in the two sexes.

Clinical History.—As has been stated, an ulcer involving the upper part of the duodenum may give rise to many and painful symptoms, whereas one located lower down is sometimes surprisingly latent. When the lesion is in the neighborhood of the bile papilla it may from obstruction set up a train of biliary symptoms.

Hall of Denver reports having seen cases with typical attacks of gall-stone colic, jaundice and tenderness over the gall-bladder occasioned by peptic ulcer the edge of which involved the outlet of the common duct.

It is not very unusual to find cholecystitis and duodenal ulcer coincidently. When the disease extends to the pancreas, as it often does, symptoms of chronic pancreatitis are encountered. George Herschel 20 finds Cammidge's "C" reaction to be a help in the diagnosis of duodenal ulcer, owing to the fact of pancreatic involvement. Of course the pancreas may be invaded by gastric ulcer when this arises on the posterior wall and extends pos-Einhorn reports a careful study of six cases of duodenal ulcer recognized by his string test, and in some instances verified by operation. In all six there was marked hyperchlorhydria, in two a slight continuous secretion and in one peristaltic unrest. Each of these cases showed periods free from symptoms alternating with periods of distress. Hemorrhage occurred in two cases only. There was constipation in four and looseness of the bowels in two. Tenderness to pressure was absent in all but one case. Pain appeared between two and three hours after taking food and was relieved by

<sup>20</sup> Clin. Jour., Jan. 12, 1910.

eating. This is the characteristic "hunger pain" of Moynihan, a symptom which many surgeons have emphasized. While it undoubtedly occurs in most cases of duodenal ulcer, so it does in pyloric ulcer, and in neurotic individuals it is a common accompaniment of hyperchlorhydria when no lesion of the stomach exists. I have observed this pain, in some patients known to have duodenal ulcer, within an hour after meals; and in others "hunger pain" was continuously absent. There is a tendency to exaggerate this symptom as a criterion in differential diagnosis. It is noticeable that looseness of the bowels, an unusual symptom in gastric ulcer, is frequently present in duodenal ulcer.

Gastric motor insufficiency, so often present in ulcer of the stomach when situated near the pylorus, also occurs in some cases of duodenal ulcer. In certain cases the stomach empties itself hurriedly, especially of a bismuth meal.

In exceptional cases the pain of duodenal ulcer is not experienced in the abdomen but above the diaphragm. At times, but not always, this is to be explained by pleuritis occurring as a complication.

Perforation.—The symptoms of perforating duodenal ulcer are for the most part identical with those of gastric perforation. Some cases are latent to a remarkable degree and the first intimation of the disease is the onset of the signs and symptoms of perforation.

When perforation occurs on the posterior wall of the duodenum, it may give rise to no immediate symptoms or only to those that are so mild and indefinite that their nature is not suspected. Subsequently there develops in these cases manifestations of retroperitoneal abscess and symptoms of septicemia. The signs of acute perforation change within a few hours. At first the abdomen is flat,

there is the usual picture of abdominal shock. Then there develops distension of the abdomen and often there is a decline in the severity of the pain and other symptoms. This is particularly true when morphin has been given. The apparent improvement in the patient's condition may lead to fatal delay in operation. Generally there is a rise in the white blood count and in the blood pressure, but too much reliance should not be put in any one sign or laboratory test.

Some cases of lobar pneumonia and acute pleurisy mark their onset by intense pain and tenderness, and rigidity in the right upper quadrant of the abdomen. Such cases have been mistaken for perforating ulcer. The occurrence of a rising leucocyte count serves to further the misconception. A decrease of hepatic dullness is not a reliable indication of perforation.

#### DIAGNOSIS

Since the days of Cruveilhier, the diagnosis of peptic ulcer has been based upon three cardinal symptoms: pain, vomiting and hemorrhage. These continue to be the symptoms of greatest importance. Sometimes one or another of these fails to appear. The occurrence of hematemesis was formerly thought to clinch the diagnosis, but The diagnosis should be made without not so to-day. waiting for hemorrhage, and we should remember that hematemesis often occurs without ulcer. The importance of vomiting as a symptom has been exaggerated. It probably occurs in less than 50 per cent of cases even when they are advanced. The most important symptom is the localized pain, especially when it takes an anteroposterior direction and when it is accompanied by localized tenderness both in the epigastrium and in the tenth or eleventh left interspace near the spine. Persistent hyperchlorhydria is an important symptom. Tendency toward either stasis or gastrosuccorrhea are indicative of ulcer in the pyloric region.

Of very great value is the finding of occult blood in the stomach contents and stools of patients who are without other sources of bleeding in the alimentary tract, and who for two days have not eaten meat.

An early diagnosis of gastric ulcer may be made when there is pain and tenderness definitely located, when the distress increases with the gastric tension accompanying digestion, when there is hyperchlorhydria, when the string test is positive, and when occult blood is continuously present.

Pain which occurs at the moment of eating or directly after a meal is characteristic of ulcer at the cardiac end of the stomach. Early, though not such immediate and severe pain occurs with ulcer located at the fundus or at the beginning of the lesser curvature of the stomach. Pain may be early when the disease is at the pylorus, but more often pyloric ulcer, like duodenal ulcer, leads to pain two or three hours after a meal. Prompt relief of pain after the administration of 1 gm. (15 gr.) of orthoform, as suggested by Murdock, is strongly indicative of ulcer of the stomach. Wagely has found that the pain in ulcer is promptly excited by the giving of full doses of nitrate of silver or a solution of ferric chlorid. Others, for the same purpose, have used full doses of hydrochloric acid. These latter measures are not as reliable as the orthoform test.

Experience with Einhorn's string test has satisfied me of its value. (See Page 77.)

# **DIFFERENTIAL DIAGNOSIS**

The diagnosis of ulcer is easy when the symptoms are typical; at other times it is difficult and occasionally impossible. Gastralgia.—Gastralgia associated with hyperchlorhydria is easily mistaken for ulcer. Intense gastralgia recurring at intervals during a long time usually depends upon syphilitic lesion of the spinal cord and may be differentiated by the Wassermann reaction and by the presence of other manifestations of lues.

Chronic Cholecystitis and Cholelithiasis.—Chronic cholecystitis and cholelithiasis through reflex irritation, may give rise to symptoms that closely simulate ulcer. In a certain group of cases pain is experienced only in the pit of the stomach, and there is often a disappearance of the pain on the occurrence of vomiting. These cases also often show hyperchlorhydria and through the intense congestion of the stomach, which is increased by retching, sufficient blood is discharged to give a positive occult blood test. . Differential diagnosis may be made from the fact that in cholecystitis there is generally some evidence of sympathetic disturbance of the liver. Moreover, there may be found the tongue-like, downward projection of the right lobe, the so-called Riedel's lobe, and there is usually found a lack of clearness or a subicteric appearance in the skin. Usually definite tenderness may be elicited upon deep pressure just under the free border of the ribs, on a line extending between the right nipple and the umbilicus. Tenderness is also usually present about one inch from the vertebrae, somewhere between the tenth and twelfth intercostal spaces on the right side. There is absence of tenderness over the tenth or eleventh spaces on the left side; that is, at the point characteristic of ulcer. If the patient is carefully studied, there will be found, as a rule, occasional slight elevations of temperature, accompanied by general hebetude, loss of appetite and coated tongue. At such times, we are likely to find an increase in the white blood count. There is often urobilin in the urine.

Chronic Appendicitis.—Chronic appendicitis sometimes masks the symptoms of peptic ulcer. I refer to cases in which there occur epigastric pain and tenderness, nausea and vomiting, sometimes hyperchlorhydria, and distress or pain, soon after eating. It should be noted that pain in chronic appendicitis, as in cholecystitis, is likely to radiate through the right rather than the left side. This is generally although not uniformly the case. The pain of gastric ulcer, though it may extend to the right side instead of the left, even reaching to the right shoulder or the right intercostal nerves does so only exceptionally. As a rule, the pain of gastric ulcer, when it extends beyond the epigastrium, is directed toward the back and upwards, to the left intercostal nerves to the precordium and even to the left shoulder. It rarely extends downward into the abdomen save when there are complications. In chronic appendicitis and to a less extent in cholecystitis, there are found symptoms of intestinal irritation, and when there is evidence of marked disturbance of the colon, we may acquit gastric ulcer of being the offender, although duodenal ulcer may occasion diarrhea. It is hardly necessary to say that in appendicitis we almost invariably find characteristic tenderness over McBurney's point, yet the sympathetic ganglia also show tenderness. Differentiation when difficult may become possible by use of the thread test.

Renal Colic.—Renal colic is not often mistaken for the pain of gastric ulcer, but doubt is occasionally felt in differentiating between it and perforating ulcer. In severe cases of nephritic colic the pain may be located in the epigastrium; there may be abdominal shock and at times syncope with vomiting. By examination of the urine and a discriminating analysis of the symptoms and signs, ulcer may be excluded.

Floating Kidney.—Floating kidney, giving rise to a twist of the ureter, may excite pain of severest character, a condition known as Dietl's crisis. This occurs in the left kidney especially, when it may be mistaken for perforating ulcer. Such an attack arising in a person having hyperchlorhydria might be misinterpreted.

Case No. XVII.—A trained nurse, a powerful woman of middle age, suffered abdominal shock and terrific pain. She was at times almost pulseless with periods of syncope. Vomiting was persistent, the vomitus blood-streaked and there was exquisite, rather general, abdominal tenderness. The pain was mostly epigastric and did not radiate down the ureter. However, a diagnosis of floating kidney was made. Dr. Park performed the operation of fixation of the kidney and the patient was cured. Strange to say, the patient two years later developed identical symptoms, this time related to the right kidney. When this was anchored the patient was cured and remained well.

Torsion of a pedicle, internal hernia and embolism of a mesenteric vessel may induce symptoms that suggest perforation.

Gastric Erythism.—Gastric erythism is a term used to designate a condition fortunately rare, which is sometimes with difficulty differentiated from gastric ulcer. What was considered a typical case is here described:

Case No. XVIII.—A Professional Colleague Had Pain, Hematemesis and Hyperchlorhydria. Exploratory Operation. No Ulcer. Bleeding of Mucosa. There was a definite area of tenderness, hyperchlorhydria, vomiting preceded by eructations, heart burn, and occasionally severe hematemesis. He was always temporarily relieved by the accepted treatment for gastric ulcer, and was free from suffering when he lived an active out-of-door life undisturbed by professional re-

sponsibility. As soon as he returned to his duties as teacher and practitioner, the painful symptom-complex reappeared. An exploration was made by Dr. Park in the presence of several other surgeons. Not a vestige of ulcer could be found, although the stomach and duodenum were minutely and thoroughly searched, but there was discovered an extraordinary condition, embracing the entire gastric mucosa, but somewhat more marked toward the pyloric extremity. There was great turgescence of the mucosa and blood oozed freely from all parts of the mucous surfaces upon the slightest manipulation. However, no break in the mucosa could be discovered. even by the use of a magnifying glass. The patient made a good recovery from the operation, but has been compelled to relinquish his professional career and retire to a farm. Now that he leads the life of an agriculturist he is able to eat without restriction and is entirely free from his symptoms. I have collected the histories of a series of cases which correspond closely with the above.

A new use of the duodenal tube is that of "autodrainage." Writing from von Noorden's Clinic to Archiv. fur Verdauungs-Krankheiten (abstracted in Jour. Am. Med. Assoc., Jan. 24, 1914), S. Bondi describes a method of utilizing the duodenal tube that promises to be useful.

The bucket having reached the duodenum, the patient lies on the right side, the end of the tube hanging over the edge of the bed, whereupon the duodenal fluid drips into a receptacle. By observing and testing the discharge from time to time variation in the character of the fluid may be noted. Intermittent flow of bile, the presence of blood, pus cells and biliary casts, the color and the reaction may thus be discovered; this method may materially assist in differential diagnosis.

# CHAPTER XII

# PEPTIC ULCER Continued

#### TREATMENT

In stating the nature of peptic ulcer, in the description of its morbid anatomy, pathogenicity and clinical course, as well as in the brief allusion made to its complications, I have had in mind the indications for treatment. This is particularly true as regards the disturbed physiology which, it is believed, is largely responsible for the existence of the disease as well as for its continuance, exacerbations and complications. The treatment of some of the complications requires special consideration and in another chapter will be given amplication. The therapeutic indications naturally fall into divisions, depending upon the several morbid activities which go to make up the pathology of this gastric disease.

- (1) Hyperacidity, hypersecretion and irregular secretion.
- (2) Motor excitability, gastric unrest, spasm, obstruction, vomiting and stasis.
  - (3) Autodigestion, loss of substance and hemorrhage.
- (4) Involvement of the serous coat and contiguous parts.

It is evident that the therapeutic indications for the relief of symptoms arising from one of these groups of phenomena often merge into those of another group, but for the sake of clearness in statement, the attempt will be made to consider them, as far as possible, separately.

Measures that are merely empirical are pushed aside with the aim of satisfying definitely the demands of therapeutic indications.

# INDICATIONS FOR TREATMENT OF HYPERACIDITY, HYPERSECRETION AND IRREGULAR SECRETION

Hyperacidity and over-secretion result either from local irritation excited by the development of the ulcer, or from general or local nervous disturbance.

When a case of recent ulcer presents itself, careful inquiry should be made as to whether the attack began abruptly or whether it was preceded by a long period of irritative stomach symptoms.

In the latter event, it is probable that the case began with hyperchlorhydria, resting on ground other than that of ulcer, and that the hyperacidity played a part in the evolution of the ulcer. It is possible that the element of over-acidity and over-secretion may occasion pain and vomiting without the occurrence of spasm or obstruction. Indeed, sometimes the stomach under these circumstances empties itself quickly, surpassing in this respect the normal stomach. Under such circumstances, it is quite conceivable that the motor function is over-stimulated, but that thus far it has worked in an orderly manner. The vomiting may be the result of irritation engendered by the hyperacidity, or, together with the pain, may partly depend upon hyperesthesia.

Hyperesthesia.—A zone of hyperesthetic tissue may precede as well as accompany recent ulcer. It is because of this hyperesthesia that pain and vomiting may ensue in cases in which the acidity is not above the usual point or even where there is subacidity. In order to understand the therapeutic indications, it is important to bear this in mind. When hyperesthesia is present, the stomach be-

comes resentful even of a comparatively low acidity and comfort is not obtained without the use of antacid and other soothing measures. Antacids are needed in these cases in proportion to whatever relief they give, and we need not hesitate to prescribe them even though the laboratory reports a low acidity in the gastric contents. In selecting the remedy, it is well to consider that while we are aiming to neutralize an acid, we are also attempting to sooth a hyperesthetic part. This explains the fact that a mixture containing cerium oxalate or one of the bismuth salts is more efficacious than is light magnesium carbonate or sodium bicarbonate alone. In certain cases greater relief follows the administration of large doses of bismuth, 1 to 4 gm. (15 to 60 gr.). Benefit may be obtained by the use of nitrate of silver. After its first stimulating effects, there sometimes ensues a state of comparative calm. Although silver is advocated by Rokitansky and his followers, I find it inferior to other measures and rarely employ it. The various preparations of phenol are occasionally indicated and may be given in the following mixture:

 Phenolis
 2.00 (3 ss)

 Mucilaginis altheae
 Aquae laurocerasi
 aa 60.00 (3 ii)
 M.

 Sig.—A teaspoonful every two hours.

This is not to be continued for more than one or two days at a time.

Iodoform with equal parts of bismuth in doses of .06 to .20 gm. (gr.i—gr.iii), three to four times daily, given in a capsule is often useful. I have found decided relief in some cases from ichthyol, 3 to 5 drops, blended with an equal amount of castor oil, given in a capsule, half an hour before taking food; or the proprietary preparation known as Lignol may be given in like dose and in the same manner.

Prompt relief of pain and other symptoms depending on hyperesthesia is produced by the taking of small doses of opium or its derivatives. Formerly I was in the habit of prescribing "old opium pills" which contained ½ gr. of opium, and which were to harden by age so that dissolving slowly, they might exercise a rather slow and continuous local effect. Of late years I have omitted opium almost entirely in the treatment of ulcer. First, because it is found to be unnecessary, and, second, because of the deplorable cases of opium addiction brought about through its use in this disease. When, as is usual, there is hyperchlorhydria or over-secretion associated with the hyperesthesia, belladonna, hyoscyamus or their alkaloids are less objectionable than opium. Even these drugs should only be employed when simpler sedatives and antacids fail to produce relief. In those cases in which suffering continues, notwithstanding the intelligent use of bismuth and the various antacids, belladonna proves to be very service-One should begin with small doses so as to avoid the untoward effects of the drug; that is, dryness of the buccal and nasal mucosa, mydriasis and cutaneous irrita-The beginning dose may be as small as 2 or 3 drops of the tincture, given in a tablespoonful of water three times daily; it should be increased regularly drop by drop, until relief is obtained or until the unpleasant physiological effect of the drug indicates a decrease in dosage. I sometimes give the remedy in the following prescription:

Extracti belladonnae fluidi ................................. 1.25 (m xx)
Tragacanthae
Lycopodii aa q.s. M.
Divide into forty pills.
Of these one or more may be taken three times a day.

When it acts happily, belladonna undoubtedly results in decreasing secretion as well as relieving spasm and hyperesthesia. We should remember that in very early cases of gastric ulcer, an inflammatory reaction is developing at the border of the ulcer, a factor that is more important in some cases than in others. If we can succeed in overcoming this inflammation or confining it to narrow limitations we shall have greater benefit from the application of the antacids and other sedatives. Therefore it is recommended to try frequent small doses of the following prescription:

Tincturae aconitae, U. S. P 1.00	(m. xv)
Tincturae belladonnae 2.00	(3  ss)
Antipyrinae 2.00	(3 ss)
Aquae aurantii floridis q.s. ad60.00	(3 ii)
•	` M.

Of this a teaspoonful is given every two hours until the pain is subdued. With this an antacid should be employed. Should it happen, as is occasionally true, that bismuth and magnesium are unsatisfactory, the officinal lime water, in dessertspoonful doses, well diluted, taken every half hour or hour, gives a gratifying result. Comfort may follow the use of minute doses of the acetate of lead. However, its continued employment has objections that need hardly be mentioned. In one case I observed unexpected but remarkable benefit from the frequent taking of two or three drops of eucalyptol. Camphophenique, diluted with castor oil or combined with ichthyol, is at times of distinct advantage.

The following prescription, having the combined effect of these drugs is useful in ameliorating the distress occasioned by the gastritis.

Ŗ	Eucalyptolis	0.12	(m. ii)
	"Camphophenique"	0.12	(m. ii)
	Ichthyolis,		
	Olei Ricini		

M.

To be blended and taken in a capsule about once in four hours.

In the hyperesthesia of ulcer where pain is an important and incessant symptom, an effectual remedy in some patients consists of chloroform in conjunction with bismuth.

Chloroform water may be given alone, or combined with a modicum of menthol, for the relief of pain or of burning distress that presages paroxysms of pain.

There should not be over-readiness in relieving pain without pausing to question its origin. There must be a reason for the pain and its source should be inquired into while attempting its relief.

Are we sure that the factor of over-acidity is fully antagonized? Are we satisfied that the local tension incident to circumscribed gastritis and over-tonus of the organ as a whole has received due consideration? If not, then, in the first instance, let the alkaline medication be followed more thoroughly; in the second instance, let more attention be devoted to subduing tension and inflammation by procuring complete rest of the stomach for a day or two by giving small repeated doses of aconite. antipyrin or aspirin, by applying ice continuously over the epigastrium, or by the more assiduous application of hot compresses. Thus by exercising discrimination we may avoid the use of measures that act mostly to obtund sensation, and in no way retard the progress of the ulcer. The aim should be to relieve symptoms, not by the use of anodynes, but by correcting the disturbed physiology. This course may assist in curing the ulcer as well as relieving distress. By the use of analgesics we may delude ourselves into believing that the patient is improving, although in fact the erosion and the gastritis are advancing.

These prescriptions are suggested not because they constitute the most commonly useful remedies in recent gastric ulcer, but in order to emphasize the fact that hyperesthesia and beginning gastritis may need special treatment and that by resorting to such measures the general plan of treatment may be made more easily successful and may need to be prosecuted with less energy.

In the majority of cases, in addition to rest and dietetic measures, all that will be required is the administration of a combination of cerium oxalate, bismuth subcarbonate, and magnesium carbonate or light oxid. After the study of many combinations I find the following prescription to be the most satisfactory:

Ŗ		
·	Cerii oxalatis	two parts

Of this half a teaspoonful or a teaspoonful whipped up briskly in a wine-glassful of water should be taken every two to four hours. Or in case the symptoms are acute, it may be repeated every half hour for three or four doses and then omitted until there is a suggestion of the return of symptoms. Sometimes this mixture is not sufficiently alkaline, in which case it may be made more efficacious by the addition of four parts of sodium bicarbonate, or the latter may be given in doses of half a teaspoonful dissolved in half a glass of water, alternately with the foregoing prescription. Large and repeated doses of sodium bicarbonate are contraindicated and may prove to be harmful. With gastric ulcer there is usually constipation, which is overcome by the magnesium in the above prescription. If the magnesium induces diarrhea, it is

best to substitute the subgallate for the subcarbonate of bismuth or the following:

Ŗ		
•	Cerii oxalatis	
	Bismuthi subcarbonatis	two parts
	Cretae preparatae	•
	Sodii bicarbonatisaa	four parts
		<b>M</b> .

Of this a teaspoonful thoroughly mixed with a claret glass of water may be taken every two hours until looseness of the bowels is controlled, whereupon one should return to the former prescription. The constipation above referred to is sometimes a troublesome symptom and is not perfectly relieved by the magnesium mixture, the socalled "gastric sedative." In such an event, there should be given each morning a saline draught in sufficient amount to produce the desired result. In Germany Carlsbad water is largely in vogue for this purpose. It has seemed to me that better results are obtained by the use of a saline containing less sodium chlorid. Rochelle salts given early each morning acts satisfactorily and is free from the objection just mentioned. Aside from the careful administration of these simple remedies, there is little need of further medication in cases of recent ulcer. a condition in which physiological therapeutics is of paramount importance, and this may be summed up in the term physiologic rest. This means not only rest of the organism, but rest of the stomach as well, and in saying rest of the organism, something more is meant than is usually understood by "putting the patient to bed." Rest of the body in the horizontal position is a sine qua non of treatment, but, in addition to this, steps must be taken to procure complete mental rest as well. fore it is most advisable to have the patient removed to a hospital or sanitarium where he can escape from the

mental and moral stress which is entailed by illness in almost any household where the patient is surrounded by the interests, duties and responsibilities making up the average life about him. The inadequacy of the rest treatment at home, the patient can hardly realize until he has experienced the relief which attends isolation in a proper institution. If these simple measures were perfectly understood and the clear indications acted upon intelligently there would be fewer cases of recent ulcer accompanied by calamitous events and fewer that would develop into chronic ulcer.

It is next to be insisted upon that the rest which is here described should be continued for a sufficiently long time. In the average practice a victim of gastric ulcer is discharged from treatment, and especially the complete rest is relaxed, before the ulcer is healed and before the disposition to recur has passed away. Bettman of Cincinnati has clearly voiced this truth, and I am convinced that if the principles here set down were conscientiously carried out there would be found far less need of surgical intervention in gastric ulcer. No clinician in family practice can fail to be aware of the embarrassment which awaits the man who has the foresight and the determination to outline and carry out the proper course of treatment in this disease. It is not that the physician is lacking in knowledge of what is necessary, not that he is indifferent to the safety and the ultimate welfare of the patient, but because he is borne down and turned aside from the course which his judgment dictates by the pernicious opposition, the stupidity, ignorance and superstition of the patient, or his family and friends. Of course this is true with other diseases besides peptic ulcer. some communities it is more true than in others, but in nearly all diseases and nearly all communities, the physi-

cian has a burden to bear in this respect. Yet, onerous as this burden is, I cannot help making this plea for greater rigor in the exercise of unquestionable duty in the management of all cases of recent gastric ulcer. Occasionally we shall be thwarted through obstinacy of the patient, as I was in illustrative case No. XII. That case occurred many years ago, before my present views were so definitely fixed, before I had seen so many direful results following blundering and careless management of the disease and before I had realized that in the practice of medicine there must always be someone who commands, either the patient and his family, or the physician in charge. When the former are at the helm, the voyage is apt to be long and tempestuous, and very liable to terminate in disaster. When the physician really takes command there probably will be an enormous economy in suffering, risk and expenditure of money and time.

To resume, let it be repeated that the essential thing in the treatment of peptic ulcer is physiologic rest in the widest interpretation of the term. The patient should be isolated to that extent which induces the greatest calm. It is to be expected that the pain will quickly subside and that the seclusion and the restricted diet will soon prove to be the greatest strain upon his self-control. fore it is the part of wisdom so to surround him that he may be at peace. The light and ventilation of the room, the position of the bed in relation to the window and particularly the comfort of the bed are not too humble matters for our care. A daily sponge bath should be given. and, where it can be afforded, passive massage each day by a skilled operator, avoiding of course all manipulations of the abdomen. It may be objected that this is not rest, which in a sense is true, and it is true that when the masseur is lacking in skill or in personal tact he will increase the arterial and muscular tension through irritating the nervous system, in which case the patient will feel distinctly worse as a result of the treatment. On the other hand, in patients accustomed to an active life, a state of restlessness ensues after a short confinement in bed, and this may be notably relieved by a skilled masseur, who knows how to soothe and relax his patient. Sometimes I have thought that a deaf-mute would be the ideal person to give such treatment. When there is neuralgia secondary to ulcer, when there is pain radiating into the intercostal region and extending to the spine, great relief will follow spinal nerve pressure with slight vibration. This, even to a considerable extent, may relieve the epigastric pain. Within the circle of physiologic rest is included the question of gastric rest, and therefore of dietetics.

In certain cases, physiologic rest of the stomach is best obtained by the practice of duodenal alimentation as devised by Einhorn. By this method, elsewhere described, the patient may be sufficiently nourished while the stomach remains undisturbed. In exceptional cases, in which pyloric spasm is intense, there may be delay in the passage of the tube into the duodenum; yet it is in these cases that the method is most serviceable. With a display of confidence, the practice of patience and perhaps an injection of atropin, success is usually obtained. The beneficial results are remarkable, and this method is likely to supersede older forms of medical treatment and to obviate the need of surgical intervention in the majority of cases in which it is now practiced.

Before taking up the question of diet it will be more convenient to consider the second division of the indications for treatment.

#### INDICATIONS FOR TREATMENT OF MOTOR EXCITABIL-ITY, GASTRIC UNREST, SPASM, OBSTRUCTION, VOMITING AND STASIS

Certain individuals possess naturally a tendency to motor excitement. In such people comparatively slight causes lead to spasmodic contraction. Familiar examples are ticklishness, a tendency to sneeze, cough, yawn and strangle from trivial causes. In such individuals, an over-motility of the intestine is easily induced, so that diarrhea or at other times spastic constipation may develop. It is therefore natural to expect that certain patients would suffer more than others from motor excitability of the stomach. The swallowing of a dry crust, the over-stimulation from condiment, or undiluted spirits is in these individuals followed by spasm of the cardia, or gastrospasm with very considerable discomfort. der the circumstances it is to be expected that high gastric acidity would produce disagreeable motor symptoms and with the advent of gastric ulcer, the element of spasm would become prominent.

Measures which decrease the stimulation of the gastric mucosa will decrease the motor excitability. It is because of this that the treatment hitherto described for the abatement of the over-acidity will have a calmative effect upon gastric motion. One is not always able to decide whether the pain complained of is occasioned by the acidity alone or by the spasm which acidity induces. Pain almost invariably develops as a result of spasm or of the over-tonicity which precedes it. At times we infer that the pain results from high acidity without spasm; in other cases it is quite clear that the pain comes only when spasm develops. At any rate when there is spasm we should be particular in our efforts to minimize gastric acidity. There is no one measure so immediately suc-

cessful in this as lavage, which results in thoroughly cleansing the stomach. Fasting after lavage should be prescribed until the motor excitement has quieted down. This is especially true when the ulcer is near the pylorus. When the ulcer is near the cardia, lavage is often distressing and has to be dispensed with. Then we must resort to treatment by a period of fasting, with the exception of allowing demulcent drinks. Rectal alimentation should be practiced. The giving of frequent small doses of orthoform or anesthesin affords relief by obtunding sensibility of the exposed nerve ends; frequently doses of a mixture of bismuth in gelatine water or sips of the milk of almonds give comfort by providing a temporary protection to the eroded part. When the ulcer is at the pylorus or along the lesser curvature, lavage carefully practiced serves an admirable purpose. For a long time there was felt a timidity in the use of the stomach tube in dealing with gastric ulcer, but we have learned that this hesitation is often unjustified. Unfortunately people who suffer from an unusual motor excitability are often bad subjects for lavage, and it is found that the straining and gagging attending the practice does more harm than good. Whether lavage is carried on satisfactorily or not depends very much upon the tube that is selected for the purpose and upon the dexterity and the mental qualifications of the physician who uses it. Granted that the physician is expert and the tube suitable, very much can be gained by lavage. should be taken not to overdistend the stomach by using too much water at a time, and it must be so maneuvered that air is not carried into the stomach with the wash water. The temperature of the water should be that of the body. A normal saline solution or a weak solution of boric acid should be employed. The lavage should be

practiced quickly, therefore a tube of large caliber should be selected. Before withdrawing the tube, there may be introduced into the stomach a large amount of bismuth, stirred with water or with a demulcent solution. Fleiner. following the example of Kussmaul, recommends the introduction through the stomach tube, after lavage, of 70 or 80 grams of bismuth at one time. Some believe that bismuth forms a coating over the eroded surface, and through the protection thus afforded allays the irritation and consequently the spasm. Matthes, among others, claims that the bismuth has a predilection for the ulcer surface and that it is retained there after other portions of the mucosa are clear. This is said to have been demonstrated by experiments upon animals, and Hemmeter asserts that he has shown this by the use of the X-ray. There is no doubt of the soothing effect of these large doses of bismuth and the practice seems to be followed by no unpleasant consequences. Toxic effects have been reported, but these seem to have depended upon impurity in the salt employed. Some specimens of subnitrate of bismuth are found to contain toxic properties, but the subcarbonate escapes this objection. Marked constipation rarely results from this treatment. Therefore, provided the measure proves efficacious in a given case there is no reason why it should not be employed.

Pylorospasm is sometimes relieved by iodoform and belladonna and also, in certain cases, by use of the nitrate of silver as practiced by Gerhardt. He gave from .20 gm. to .30 gm. (gr.iii ss—gr.v) dissolved in 120 c.c. (3iv) of water, taken in tablespoonful doses during the day. The late William Pepper strongly advocated the use of silver nitrate in gastric ulcer.

In many cases the element of spasm becomes obstinate,

apparently as the direct result of hepatic congestion. I have convinced myself that this is a reality. The patient may be comfortable and free from over-tonicity and over-contractility until suddenly increased pain and other symptoms of spasm, including stasis, show themselves. If careful physical examination is now made it will be found that there is unusual resistance over the lower end of the stomach, and that the liver is moderately enlarged and somewhat tender to palpation. Under these conditions there should be given a single large dose of calomel, .30 gm. (gr.v) and followed by 30.00 gm. (3i) magnesium sulphate dissolved in 250 c.c. (3viii) of warm water. There should be expected a thorough emptying of the intestine by this means, a recession in the size of the liver and a subsidence of the spasm, sensitiveness and hyperacidity. Constipation must be avoided in cases in which pyloric spasm is a prominent condition, and it is for this reason, among others, that the use of Carlsbad water as a routine measure has achieved its reputation. I have given my reasons for preferring other salines, but it cannot be denied that the use of Carlsbad water has won approval of some of the best clinicians, especially the Germans. However, apparently as good results, and it seems to me even better, follow the use of Rochelle salts, sulphate of magnesia, castor oil or phenolphthalein.

In some cases of pyloric spasm, even when caused by duodenal ulcer, I have found that saline purgatives produce gastric distension, pain, vomiting and marked general depression, yet without the desired purgative action. This, perhaps, has explanation in the experimental work of Brown of Johns Hopkins, who found that hypertonic solutions of saline purgatives are not absorbed until through active gastric secretion they become isotonic

with the blood. They do not pass the pylorus until the solution is isotonic. These facts in connection with some bad clinical results which I have had, make me reluctant to employ saline purgatives when pyloric spasm is an important feature in peptic ulcer. Indeed, high enemata are more satisfactory than any purgative except in case it is desirable to excite secretion from the upper digestive tract.

Of all measures for the prevention and relief of motor excitability of the stomach there is nothing so free from objection and so generally useful as the continued application of large hot fomentations or poultices. It will be remembered that this constitutes an important feature of the von Leube method of treatment. There are many reasons why these local applications should be adopted as a routine measure in the treatment of peptic ulcer. They assist to bring about general relaxation of the patient, probably because they soothe the abdominal sympathetics, and they are very successful, not only in . preventing gastric spasm, but in relieving that condition when it has once developed. Another reason for advising the hot stupes or poultices is that the skill and attention required for their proper application offer an additional explanation to the patient for his lying at rest and having the benefit of a trained attendant. I have found it easier to restrain patients upon whom poulticing is practiced than others not similarly treated. Von Leube points out the importance of the careful preparation of the surface of the abdomen before and during the poulticing. Unless much care is shown, the skin becomes softened and may become infected. Therefore it is advised to shave the surface of the abdomen, to apply often some unirritating antiseptic lotion and to anoint the part with lanolin cream or vaselin. It may be found

advisable to continue the application of the poultices for several weeks in succession. Ordinarily ten or fourteen days is sufficient; should spasm and soreness persist a longer course is advisable. When there is hemorrhage, the hot applications should be relinquished, and the ice bladder substituted until bleeding ceases; later iced, wet compresses, after the method of Winternitz, should be applied.

When vomiting becomes a prominent feature, the local application of ice is preferable to hot applications. Vomiting may be combated by administering repeatedly large doses of cerium oxalate, .30 or .60 gm. (gr.v—x) or by giving one drop doses of tincture of ipecac every hour, or .30 gm. (gr.v) doses of orexin tannate, or by the temporary use of the following prescription:

R <sub>2</sub>	
Cocainae hydrochloratis 0.06	(gr. i)
Phenolis	
Mucilaginis altheae 8.00	(3ii)
Aquae laurocerasi vel aquae chloroformi30.00	(3i)
	M

Sig. One teaspoonful every half hour for a few doses or until the vomiting is controlled.

Dilute hydrocyanic acid, in doses of 0.12 c.c. to 0.30 c.c. (m.ii—m.v) is useful in controlling vomiting.

Occasionally, when there is marked local irritation, vomiting may be relieved by purified liquid vaselin, white Russian oil, liquid paraffin. This substance is also of use in relieving pain and motor excitability and I frequently recommend it in doses of from 8.00 c.c. to 15.00 c.c. (3ii to 3iv), t.i.d., as a routine treatment. Sir Arbuthnot Lane regards this remedy as the most curative of ulcer.

### INDICATIONS FOR THE TREATMENT OF AUTO-DIGES-TION, LOSS OF SUBSTANCE AND HEMORRHAGE

As a rule we are first apprised of the extensive loss of substance through deep auto-digestion by the appear-

ance of hemorrhage This occurrence greatly alarms the patient and family and is for this reason not without its advantages, because it makes less troublesome the carrying out of necessary discipline in the case. Hemorrhage is not usually alarming to the experienced clinician for he has learned that with proper treatment hemorrhage is generally controlled. I have several times had the experience of exciting acute hemorrhage by the use of the stomach tube in case of gastric ulcer. At first this led me to fear this consequence of lavage, but further experience has served to dispel this timidity. Now, when hemorrhage develops. I do not remove the tube, but substitute ice water, according to the recommendations of Ewald, for the warm water usually employed in lavage. This measure usually suffices to stop the hemorrhage; but I am not content with this for I wish to feel sure that the involved vessel will remain closed long enough for the formation of a thrombus. Therefore after the use of the ice water, I introduce a solution of adrenalin, about 1.00 c.c. of the 1 to 1000 solution, diluted in 30 c.c. of water. This is allowed to remain in the stomach for a few minutes when it is washed out and a second dose introduced in the same way and allowed to remain. This generally stops the hemorrhage, unless an unusually large vessel is open. Following this treatment, I give hydrastin hydrochlorate 0.008 (1/8 gr.) tablet form, every hour for a few doses, or 1.00 c.c. (m. xv) of the fluid extract of ergot every hour. Occasionally I find that the fluid extract of hamamelis in doses of 1.00 c.c. (m. xv), given every hour is attended with better results than ergot or hydrastin. Sometimes "styptin," 0.30 (gr. v) behaves well. Aside from these remedies, nothing should be swallowed, not even water. The patient should be put to bed, kept absolutely quiet and the routine treatment of ulcer instituted, special attention being given to hemorrhage should it recur, particularly in those cases in which the hematemesis is the primary announcement that gastric ulcer has to be dealt with. It must be repeated that perfect rest is demanded above all things in these cases, not to relieve over-secretion or spasticity but to favor thrombus formation and stoppage of hemorrhage. In order to secure mental calm and to favor physical rest, it is advisable to inject subcutaneously 1/6 gr. of morphin combined with \(\frac{1}{200}\) gr. of atropin. a measure which may be repeated as circumstances require. Ice should be applied over the epigastrium. The common practice of resorting to the use of a normal saline injection, either by the subcutaneous method or by enteroclysis, is strongly to be objected to. It merely serves to fill the blood vessels, to lessen the coagulability of the blood and to raise the blood pressure, thus favoring the recurrence of hemorrhage. I am satisfied that I have seen harm result from this measure thoughtlessly employed. It would have a possible use only in the event of loss of blood so great as to bring about danger of a fatal termination from this withdrawal of the fluid from the body, and such a state of affairs is scarcely conceivable in the early hemorrhages. Even in such a case, if the severed vessel is so large that the hemorrhage cannot be controlled in a short time by the application of the measures here considered, then surely death cannot be averted by temporarily sustaining the patient by the use of a normal saline injection. A hemorrhage so formidable as this, depending upon the rupturing of a large artery, will prove fatal unless it is controlled by surgical measures. In grave hemorrhage operative intervention has been attended with such high mortality that medical treatment is usually depended on save under special con-

ditions, such as, for instance, stenosis, or possibly when the foregoing history indicates the existence of a chronic, indurated ulcer accompanied by persistent spasm. oft-repeated small hemorrhages that sap the vitality and are rebellious to medical treatment, operation is certainly called for. The exception to this is in the instance of gastric ulcer occurring in the hemophilic. In these the hemorrhage is wont to continue as a more or less persistent leaking, even when merely small vessels are severed. At such a time the hemorrhage may not be abundant, but it goes on unabated day after day. Local styptics, ice-water lavage, adrenalin, etc., have little effect in controlling it. Very often the trouble depends upon the lack of coagulability in the blood, as may be determined by suitable tests. This state of insufficient coagulability of the blood may be merely temporary or it may be persistent. In either event we should do whatever is possible to increase the coagulability, and this may be accomplished by the administration of calcium chlorid or calcium lactate. The latter salt being less irritative is preferable, and it is well to administer it per rectum in a dose of 4 gm. (3i) dissolved in 300 c.c. (3x) of normal saline solution, three times a day. It should be administered slowly in this quantity of water which is not likely to over-distend the blood vessels and which serves a good purpose in relieving thirst. The hemorrhage may be controlled promptly by the subcutaneous injection of serum. Ordinary horse serum may be used, care being exercised in repetition on account of the dangers of anaphylaxis. Rabbit serum is less likely to produce sensitization, and a preparation of dried serum, "coagulose," originally prepared by Busch of the University of Buffalo, has been found to be efficacious and to be practically free from the evils of anaphylaxis. After the use of calcium chlorid or lactate for three or four days, it should be discontinued for an equally long time and then resumed. In the uninterrupted exhibition of this remedy the good effect is lost after a few days. Indeed, if too long continued it appears rather to increase than to diminish the coagulability of the blood. The thirst which accompanies hemorrhage requires attention. It may be mitigated by small rectal injections of normal saline. From half a liter to a liter daily should be given in small divided portions, very slowly introduced. (See Hematemesis, page 470.)

It has been supposed, although there remains some doubt in the matter, that gastric hyperchlorhydria is promoted by an abundant supply of sodium chlorid, and this has been raised as an objection to the use of normal saline solution. An isotonic solution of sodium phosphate appears to answer the purpose quite as well as that of sodium chlorid and may be substituted. Rectal alimentation is usually advisable in case of active and prolonged hemorrhage. This means of supporting the economy will receive fuller attention hereafter. At present it is enough to say that much of the benefit supposed to obtain from rectal alimentation lies in the fact that fluid is thereby supplied to the tissues. This is of definite value, provided that it is not overdone. Aside from the restoration of water the value of nutritive enemata is yet debated. When employed with perfection of detail, the clyster not too large in amount, not too often repeated and composed of substances nutritious. absorbable and unirritating, rectal alimentation is useful. Harm comes from it in producing intolerance of the rectum, disturbance of the patient and stimulation of gastric digestion, which may be brought about through the sympathetic nervous system or through psychic suggestion.

It is therefore well not to inform the patient that he is taking nourishment per rectum, and the aliment should be so administered that the patient is unable to distinguish whether he is receiving a nutrient or a normal saline enema. As a preliminary to rectal alimentation and as a daily measure during its continuance, the descending colon should be emptied by a small enema of glycerin and boric acid solution. When the rectum becomes sensitive and is intolerant of even a small quantity. of fluid, it may be soothed by the use of an astringent wash such as tannic acid 2.00 gm. (gr. xxx), water, 300 c.c. (3x). This should be allowed to remain in the rectum for a short time previous to the introduction of the nutrient enema. If this is insufficient to quiet rectal unrest, it is best to inject 20 drops of laudanum mixed with a few spoonfuls of thin starch water and allow it to remain for half an hour before the rectal feeding.

The von Leube Treatment for Gastric Ulcer.—Von Leube has formulated special rules for the treatment of gastric ulcer. The patient is required to remain in bed for ten days. On the first day the epigastrium is washed with alcohol and sublimate solution, and then a thin linen cloth spread with ten per cent boric acid ointment is applied over it for the purpose of protecting the skin from a dermatitis which may result from the continuous application of poultices.

Hot flaxseed poultices, 20 cm. by 10 cm. are then applied over the epigastrium and changed every fifteen minutes for ten hours a day. At night a cold water compress is placed over the ointment cloth. The boric acid dressing is changed once each twenty-four hours. Usually pain and soreness disappear the fifth day of the treatment. After cessation of the poulticing, cold water compresses are applied each night for three weeks,

while during the day the patient wears a flannel bandage.

Rest, in the recumbent posture for one or two hours after each meal, is obligatory during this convalescent period. No sewing, knitting, or other work requiring bending forward of the trunk is allowed. Recent hemorrhage is a contra-indication to the poulticing, and three months must have elapsed since the last hemorrhage. If hemorrhage is present at the beginning of the treatment, an ice bladder is applied to the epigastrium and is later replaced by cold water compresses.

Each morning 250 c.c. of Carlsbad water is given. A pause is to be made between mouthfuls, so that ten or fifteen minutes is occupied in swallowing the whole amount. The Carlsbad water is continued four weeks, and during the day slightly alkaline water is used to quench thirst.

If they are required for pain, gastric sedatives are given. Constipation is relieved by enemata during the ten days of active treatment and thereafter it is treated by the administration of 4 c.c. of a powder composed of 20 parts of powdered rhubarb, 15 parts of sodium sulphate and 7½ parts of sodium bicarbonate.

The diet consists of boiled milk, meat solution and softened zwiebach, during the period of poulticing. The following week, soup, sago, rice thoroughly boiled in milk, white of egg, raw and soft boiled eggs, calf's brain, and boiled chicken are allowed. Other tender meats are generally added and after the fifth week the diet of health is resumed.

Von Leube holds that the mortality of gastric ulcer is about 13 per cent; 6 per cent to 7 per cent being due to perforation and 3 per cent to 5 per cent to hemorrhage. Of 556 of von Leube's cases, 12 died; that is, a mortality

of 2.2 per cent, of which 6 died of perforation and 6 from hemorrhage. In 69 of the cases the method was not completely carried out; this leaves 424 cases of which 314 (74.1 per cent) were cured; 93 (21.9 per cent) improved; 7 (1.6 per cent) were not relieved, and 10 (2.4 per cent) died.

Von Leube claims that in from 75 per cent to 96 per cent of cases, surgery is uncalled for; and in 75 per cent surgery is meddlesome. In the remaining 21 per cent two or three courses of treatment should be given before having recourse to surgery.

Lenhartz Treatment of Gastric Ulcer.—The Lenhartz treatment of gastric ulcer consists of absolute rest in bed for four or more weeks with an ice bag over the epigastrium for the first two weeks.

On the first day 200 to 300 c.c. of iced milk are given in spoonful doses, and two to four beaten raw eggs. gm. of bismuth subnitrate is given two or three times a day for the first ten days. The beaten eggs are served from a dish packed in ice. The amount of milk is increased 100 c.c. daily and also one more egg is given, so that by the end of the first week 800 c.c. of milk and six to eight eggs constitute the daily food ration. tion, on the sixth day 35 gm. of raw chopped meat are given and the next day 70 gm., and later more if well tolerated. Rice and softened zwiebach are given about the eighth day and in the third week a mixed diet is usually tolerated. Sugar is added to the beaten eggs; beginning with 20 gm. of sugar and gradually increasing to 50 gm., and with the increase in the number of eggs; and 20 to 40 gm, of butter are given daily from the tenth to the fourteenth day of treatment. The first day of treatment the calories are 280, and the fourteenth day 3073.

Hemorrhage is not a contra-indication and Lenhartz

maintains that anemia, loss of weight and strength are in a large measure prevented by his plan.

#### DIET IN GASTRIC ULCER

In the event of hemorrhage or upon the initiation of symptoms of perforation, that is to say, with signs of localized peritonitis, the stomach should be undisturbed by food or drink of any kind. This abstinence should be practiced for 24, 48, 56 hours or longer, depending upon circumstances. The starvation cure of ulcer by withholding food and drink for two or three weeks is unnecessarv and is attended with inconveniences and risk. Among other evils there develops an acidosis of starvation that is unfavorable to the prompt healing of the tissues and is apt to induce insomnia and nervous irritability. It weakens the patient and especially in those disposed to tuberculosis may pave the way to new dangers. Rolleston has shown in the report of several interesting cases that the starvation cure of ulcer exposes the patient to infection of the buccal cavity. This is believed to result from the accumulation of bacteria in the mouth that under other circumstances are dislodged through the act of mastication. Rolleston advises, when temporary starvation is imperative, that the utmost attention be given to the cleansing of the mouth and teeth and to the frequent use of antiseptic mouth washes. In most cases it is not only unnecessary but inadvisable to require a protracted fast. The diet at first should be fluid and free from meat extracts, a restriction that holds true so long as there remains marked evidence of gastritis and hy-It should be remembered that though there peresthesia. is a definite reason for the liquid diet, no fixed date can be made for its discontinuance; it all depends upon the case in hand. This is a proper place to protest against

the routine adoption of the more formal diets in the treatment of gastric ulcer. What is said of the milk diet, the von Leube diet and the Lenhartz diet, is not conclusive; in other words, they are only useful in proportion as they are suitable to the particular case. Nothing, it seems to me, can be more inconsistent than the martinet-like exactness with which the several formal diets are carried out in this disease. The only excuse lies in the fact that certain practitioners may be more successful with a diet that is based on accepted rules than they might be otherwise, not understanding how to increase or decrease the range of food as the course of the case might indicate. When physicians feel great uncertainty as to the diet and when they are unable to secure food properly prepared, it is safer to adhere to the milk diet for the first two or three weeks and then gradually to supplement this by the addition of farinaceous substances and white of egg. It is a fact that certain persons do badly on a milk diet, probably because of repugnance to milk rather than because of inability to digest it. When the digestion seems at fault it is advisable to use a little extract of pancreas in the milk thus allowing it to peptonize slightly before being given. Lime water may be blended with milk to advantage, or vitolac, matzoon, kefir, or milk acted upon by the Bulgarian lactic acid ferment may be taken successfully. True, these preparations are acid and it might be supposed that they would add to the gastric acidity. however, is not commonly the case, unless the preparations be too old and over-acid. Lactic acid does not increase, but seems rather to diminish the amount of hydrochloric acid secreted by the stomach.

The Diet in Ordinary Cases.—It is my practice to begin the treatment in case of gastric ulcer with a milk

diet, giving about six ounces of milk once in two hours and allowing mucilaginous drinks in addition. Overkeen appetite of the patient may be mitigated by small doses of belladonna. After two or three days, the portions of milk are alternated with a like quantity of milk gruel. The farina which is used in making the gruel should be thoroughly boiled and should be prepared with culinary perfection. After two or three days of this diet there is added a thin porridge with cream and sugar, and after this hot water toast with butter, boiled rice with sugar and cream and a coddled egg.1 After ten days the attempt should be made to provide as many calories of nutriment as are necessary to satisfy the body weight of an individual kept strictly at rest and this should be our guide as to the amount of milk, carbohydrate and egg allowed per diem. When a case progresses satisfactorily, the pain and hyperesthesia having quickly disappeared under this treatment, the attempt should be made to give tender beef, mutton or chicken. At first this is a tentative undertaking and the effect upon the patient should be carefully observed. If it causes no disturbance, the meat is prescribed in the form of carefully selected steak, moderately broiled, or an equal amount of mutton chop or the breast of chicken. In case the patient is able to digest meat without reëxciting the gastric hyperesthesia, the range in food allowed is further and further extended. I have thought it inadvisable to give milk at the same meal with meat, but there is no objection to allowing well-ovened, stale bread and butter or very lightly and quickly browned toast or toasted

¹ Into a pint porcelain bowl filled with boiling water is placed an unbroken egg. The bowl is placed on the table and is allowed to stand for 8 or 10 minutes according to the size of the egg. The egg is then served and, if right, the white and yolk alike are very slightly coagulated and easily dissolved in the mouth.

crackers with meat. Fresh butter and cream are usually Fruits and green vegetables are to be well tolerated. excluded. Coffee is prohibited, but very weak tea may be allowed after the second week. When the diet has reached the above degree of liberality, the patient has probably advanced to the third week of the treatment. If he has been for a fortnight free from symptoms and if the stomach contents and the stools show the absence of occult blood, we may relax, but, nevertheless, must carefully prescribe the regimen, always being ready to give the stomach a few hours of complete rest whenever it becomes resentful of its work. A single indiscretion in diet may serve to negative all the good obtained. irritability, hyperesthesia and pain then recur, motor symptoms with spasticity develop and we have to begin all over with the disadvantage of having the patient in a worse frame of mind than at the start and the organism debilitated by the experience.

Diet During Convalescence.—When it is found that the caloric needs for normal nutrition have been satisfied through the giving of food at frequent intervals, and furthermore, when the stomach has become so quiescent that less exquisite care in feeding is demanded, measures may be adopted to provide longer periods of gastric rest. more food being given at a time and the intervals between the feedings being lengthened. The question may arise, why not resume the usual meal hours and feed three times a day? The quantity of food at one meal might then be so large as to over-distend the stomach or, at least, to put too much tension on the ulcer area. Another objection is that with the stomach left entirely without food, unless the gastric secretion has become normal, there is likely to be an accumulation of acid gastric juice in an otherwise empty stomach which, very probably,

would renew the excitement and lead to the return of symptoms. It is therefore a good plan to give solid and semi-solid food during the day in three definite repasts, with an interval of five or six hours between; then midway between these more substantial meals there should be given a cupful of warm milk or gruel. During the night the stomach should be allowed to rest, but should there occur a sense of great hunger, with "gnawing" of the stomach, this may be relieved by a tablespoonful of milk of bismuth, or a teaspoonful of the "gastric sedative" heretofore mentioned. Should this expedient be unsuccessful in relieving the gastric uneasiness, we may allow a cupful of warm milk with which is mixed a tablespoonful of lime water. As time goes on we should increase the amount of the regular meals more and more. but remain steadfast in denying all questionable articles . of diet. The patient's taste for sweets may be satisfied by allowing the simpler desserts such as ice-cream, blanc mange, sea moss farina, tapioca or corn starch, if carefully prepared, and fresh cream and sugar may be taken with them. Some patients do well on Racahout, or on weak chocolate or cocoa. The various demulcent draughts are admirably adapted for relieving thirst and soothing the sensitive stomach. The following list includes some of the best: infusion of sweet almonds,2 raisin water, infusion of slippery elm, a very weak infusion of gum acacia or flaxseed, rice, Irish moss, or gela-

<sup>&</sup>lt;sup>2</sup> Infusion of Sweet Almonds.—A handful of freshly shelled and blanched almonds. First chop fine and then grind in a mortar; to this add slowly a pint of warm water with which the almonds are to be thoroughly blended by rubbing with a pestle. Pass through a fine strainer before using.

strainer before using.

Raisin Water.—A handful of choice raisins, split with a knife and seeded, should be placed in a bowl. Over them is poured a pint of water. Allow to stand for two or three hours, after which strain.

tine. Toast water makes a satisfactory drink, and may be taken sweetened.

Rectal Alimentation.—The indication for rectal alimentation and the technique for its employment have been described. Something remains to be said as to the nature and the preparation of the aliment. The timehonored warm milk, unpeptonized, in quantities not beyond 250 c.c. is, on the whole, a safe standard for rectal feeding. Physicians have been ambitious to find a more nutritious substance without increasing the bulk, and with that purpose various substances have been blended with the milk. One of the most harmless and satisfactory is cane sugar, of which a teaspoonful may be dissolved in each milk portion, or a tablespoonful of the extract of malt (not the fermented kind) sometimes acts well. If egg is to be used, I prefer to have it peptonized and blended with 200 c.c. of very thin and thoroughly boiled starch water, rather than to combine the egg with milk. Milk is more quickly absorbed when given alone than when combined with animal proteids or carbohydrates. Fresh beef juice may be borne temporarily. too long used it is apt to irritate the rectum. It may be slightly peptonized with the extract of pancreas. know from personal experience that it is possible to sustain a patient for a considerable time by means of rectal alimentation, but the method seems to be less satisfactory in cases of gastric ulcer than in patients suffering from other conditions. This is doubtless because of the sympathetic excitement of the stomach when nutriment is introduced into the rectum. The late W. W. Potter reported many years ago a remarkable case of rectal alimentation in which the patient survived for months by this means of feeding. Nothwithstanding this, I am sure that we often delude ourselves with the belief that our patients are being properly nourished per rectum when such is far from true. It is best for us to face the fact, when we are compelled in certain cases to prescribe a fast, that the patient is really undergoing starvation and that he will probably suffer from the resulting metabolic disturbances, which we may recognize most readily by the appearance of the symptoms of acidosis. much misapprehension of the question of starvation. When driven to it we may submit a patient to complete fasting for days or even weeks without fearing a disaster, yet we must recognize that there are hidden dangers in this practice and that long periods of fasting should be avoided whenever possible. When a fast is inevitable care should be taken that a sufficient amount of water is introduced each 24 hours, and when we find that the urine is becoming over-acid, when there develops acetone and diacetic acid in the urine, we should introduce sodium bicarbonate by means of enteroclysis to minimize this acidosis.

Special formulas for rectal alimentation may be found in Chapter XXXIV.

# INDICATIONS FOR TREATMENT OF INVOLVEMENT OF THE SEROUS COAT AND THE CONTIGUOUS PARTS

When the serous covering of the stomach is irritated by advancing ulceration there is generally continuous pain, differing somewhat in character from that previously complained of. In other words, the pain of beginning peritonitis differs from that of gastric ulcer. There also develop an over-tonicity of the abdominal parietes, marked sensitiveness to pressure and all movement of the body is resented by the patient. There is usually a moderate degree of leucocytosis and a slight elevation of temperature. These premonitory symptoms of perfora-

tion are not always appreciable and may be absent; in which case, the first intimation of this dangerous feature of ulcer is of perforation itself. It is important that the early symptoms be recognized and that the parts be kept perfectly at rest in order that protecting adhesions may be formed and thus perforation avoided. development of the symptoms of local irritation or inflammation of the peritoneum the stomach must be kept as quiet as possible, the taking of food prohibited and for the most part medication by the stomach must be Morphin and atropin must be administered withheld. subcutaneously in sufficient doses to subdue gastric and intestinal peristalsis. As a rule the symptoms of peritonitis quickly subside under this method of treatment, and after a day or two the anodyne may be diminished or abandoned. Vigilant observation is called for lest acute perforation should occur under cover of the effect of anodynes. It is judicious to allow the intestinal tract to remain quiescent for two or three days and then no attempt to evacuate the bowels should be made except by the aid of simple lavement of the lower colon. use of a purgative at such time is attended with danger of separation of the fresh adhesions, thereby inviting perforation. The indications for treatment are clear. For the time being we must relinquish the treatment of the ulcer itself and concern ourselves with care of the The treatment is therefore that which we peritoneum. would adopt for localized peritonitis. It might be called the treatment of an emergency and of one of the accidents of gastric ulcer. Yet strictly speaking, the invasion of the serous coat of the stomach is a natural phase in the history of recent ulcer in those few cases in which it extends very rapidly. More often we meet with this emergency in chronic ulcer. Whenever there develop the symptoms and signs of peritonitis, a surgeon should be associated in the care of the patient so that steps may be taken to forestall perforation or to obviate loss of time in case it occurs.

#### TREATMENT OF CHRONIC PEPTIC ULCER

In the management of chronic ulcer there are involved the same principles already described in the treatment of recent ulcer, but certain questions require special consideration. Chronic ulcer as a rule has periods of quiescence and latency, interrupted by the return of acute symptoms. These exacerbations depend upon a renewal of the erosion of the ulcer and upon the reëxcitement of the localized gastritis. Therefore during these relapses we are confronted by conditions identical or analogous to those belonging to recent ulcer. At such times the patient should be submitted to the treatment for recent ulcer as heretofore described, and this treatment should be continued until we have reason to believe that the ulcer is healed. After the cessation of pain and the disappearance of minute hemorrhage, from which we may infer that the more acute manifestations have vanished. there remain not infrequently sources of irritation and obstruction for which treatment should be continued. Of course this refers especially to those cases of chronic ulcer which involve the region of the pylorus. ritation as well as the obstruction is in the inflammatory tissue which produces at the pylorus a state of rigidity and tension. This condition of the part interferes with function and when the patient attempts to resume his usual habits of life, he becomes to a greater or less degree the victim of food stagnation, and of symptoms which are occasioned by functional stress at the pylorus in its attempt, in response to an increased stimulus, to empty the stomach. Very frequently the symptoms are augmented from the fact that there remains a degree of gastritis which becomes aggravated with increased functional activity. Therefore, although symptoms belonging immediately to ulcer may be absent, nevertheless the pyloric constriction and gastritis prove very annoying. In part these symptoms depend upon the irritability of tissue and in part upon narrowing of the canal, and the stasis of the stomach contents. In most instances the latter condition is of but moderate importance so long as solids and unirritating foods are withheld. However, it is manifest that a patient cannot go on indefinitely fasting or partaking of only unstimulating pap and gruels. Something must be done to relieve the situation. We find ourselves here faced with a condition that may be called a result of ulcer, rather than ulcer itself, and the management of such cases will be considered in the following section. So far as concerns the management of a chronic ulcer, laying aside the problems more properly related to complications and sequelae, it may be summed up briefly in saying that the treatment is in principle identical with that of recent ulcer, except that it is necessarily more prolonged, the convalescence especially being protracted. The patient must be kept under surveillance for many months; the diet must be carefully selected, active work must be limited, and the patient's life generally circumscribed in its activities. Gastric lavage should be practiced sufficiently often so that we may obviate threatened stagnation and the patient must be put under still greater restriction should there develop any signs of the renewal of the ulcer. It is in cases of this kind in which ordinary existence becomes more or less a hardship and in which the patient becomes restive under necessarily protracted self-denial that the help of surgery has been enlisted. In a certain number of cases, it is the reasonable and only satisfactory means of relief.

The Question of Surgical Intervention in Chronic Ulcer.—It should be distinctly understood that reference is here made to chronic ulcer, not to the results or the complications of recent ulcer, yet in some of the latter help can only be obtained by having recourse to surgery. There are cases of chronic ulcer, especially the recurring type, in which there results neither persistent obstruction nor stasis, neither troublesome perigastritis nor serious disturbances of motion. In this class of cases, surgery is unnecessary, and the after history of such cases in which surgery has been resorted to has not in my experience been eminently satisfactory. Take for instance this case: The patient has had recurring and chronic ulcer for more than twenty years, yet during the greater part of that time under medical treatment he has been perfectly comfortable and has carried on an active business career, enjoying as much physical exercise as the ordinary business man. It is true that he has had many recurrences which have demanded a return to the treatment for acute ulcer and his convalescence has often been delayed, yet he has always recovered and he has had no more trouble than I have observed in other cases similar to his in which either drainage alone or drainage and excision of the ulcer have been performed. At one time I believed that excision of the ulcer was the rational indication. In practice this is usually followed by immediate relief, but the ulcer recurs and I am not able to say that the recurrences are less frequent than in those cases that have not been operated on.

The following is an illustrative case:

Case No. XIX.—A married woman, aged 38 and a mother, had for ten years suffered from gastric ulcer.

during which time she was scarcely ever free from symptoms. She had repeated attacks of hemorrhage and severe pain. There was no actual stasis, but there was gastric unrest, water-brash, eructations of gas and inanition which resulted from the fear of eating and from vomiting. With the hope of relieving her permanently and in the interest of economy I advised operation. There was found to be a chronic ulcer situated at the posterior portion of the lesser curvature about two inches from the pylorus. It was greatly indurated and extended into the muscular coat, but had not invaded the peritoneum. There were no perigastric adhesions, nor was there obstruction at the pylorus. The case seemed especially suited to excision, which was done. The patient made a rapid recovery and was for two years without symptoms of her disease. After the lapse of this time the symptoms of hyperchlorhydria returned, although actually the secretion was below the standard: she began to have pain and in spite of reasonable treatment at her home, she again had hematemesis. Under suitable treatment at a sanitarium she recovered and was again without symptoms for two years. Thrice since that time the ulcer has resumed activity and it has been necessary for the patient to take the cure.

It may be said that it was a mistake not to have made an anastomosis for draining in the above case. I might agree with this argument, but for the reason that I have had like experience of recurrence in similar cases in which posterior gastrojejunostomy was done. Because of these failures I am not enthusiastic as to the result of surgery in cases of chronic ulcer unattended with symptoms of stasis, or other painful and dangerous complications. In a proportion of cases, even without the complications or sequelae referred to, surgery is

indicated, so that the results of other treatment may be hastened thereby. In these cases the gastrojejunostomy does not directly cure; it merely overcomes conditions the presence of which render cure improbable. cases require subsequent medical treatment. In suitable cases excellent immediate and sometimes permanent results follow upon excision of the ulcer-bearing area. field of surgery in the treatment of ulcer is admirably expressed in a recent work by the Swiss surgeon, Pastry.4 He insists upon the necessity of medical treatment following surgical intervention in gastric ulcer. The operation per se does not cure the ulcer since it causes no transformation in the condition of the gastric walls. Operation provides the inestimable advantage in certain cases of overcoming stasis and incoercible hyperacidity, conditions that render medical treatment of little avail; but equally important is the subsequent medical care of the case, to the end that the ulcer may heal and the tendency to recur may be obviated.

### COMPLICATIONS AND SEQUELAE OF GASTRIC ULCER

Among the conditions about to be considered, some are to be regarded as the after effects or sequelae of gastric ulcer, recent or chronic, and some are to be regarded more distinctly as complications, that is, as appearing during the course of the disease. This latter is particularly true of so-called gastrosuccorrhea, or Reichmann's disease, yet it appertains also to obstruction and to other conditions in somewhat less degree.

# REICHMANN'S SYMPTOM-COMPLEX AND ITS RELATION TO GASTRIC ULCER

Without taking up in detail the time-worn subject of the etiology of gastrosuccorrhea, it may be stated unhesi-

<sup>&</sup>lt;sup>4</sup> Geor. et Cie, Geneva.

tatingly that there is a group of cases in which the typical symptom-complex first described by Reichmann finds its source in irritation at the pylorus or in its immediate neighborhood, producing exaggerated tonicity and spasm. It is not necessary that there should be food stagnation, in fact when that symptom occurs to a considerable degree, the condition is not classed as gastrosuccorrhea. The four characteristics of Reichmann's disease are the occurrence of pain four or five hours after a meal, the presence of gastric juice in the stomach even while fasting in the morning, hyperchlorhydria and finally, the rapid digestion of albuminoids. There are other features of gastrosuccorrhea which will receive consideration in a special chapter. It is enough to say here that this symptom-complex is acknowledged to arise in some cases from local pyloric disease and conspicuously from ulcer in the pyloric region. Soupault was among the first to call attention to this fact. In a considerable group of cases upon which he operated, he reports having found evidence of ulcer unexceptionally in each. Other surgeons have had similar experience, although perhaps not with the uniformity reported by Soupault. We must recognize the fact that ulcer near the pylorus, or in the upper part of the duodenum, may be masked under the characteristic symptoms of gastrosuccorrhea. The diagnostician must therefore be on his guard when he encounters Reichmann's disease against attributing it to inflammatory or neuropathic causes, when actually it may be merely the expression of local disease of the stomach. The hunger pain of duodenal ulcer is sometimes merely the result of over-secretion and relative stasis of the gastric juice which sets up this train of symptoms, embracing those of motion, secretion and sensation. When a patient who has fasted for twelve or fourteen hours,

ordinarily on waking in the morning, complains of a sensation of fullness in the stomach, when clapotage can be developed at an hour when it is normally absent, and especially when the patient complains of gastric distress and pain four or five hours after a repast, we should not omit to make a careful attempt to withdraw the stomach contents in the morning before breakfast. the passage of the stomach tube in case of Reichmann's disease there is recovered 100 c.c. to 500 c.c., or even more, of slightly opalescent gastric juice, which may have normal acid reaction or which may show hyperchlorhydria even of high degree. Morning vomiting may occur, in which case the vomitus will correspond with the gastric juice just described. How are we to determine that this symptom-complex depends upon ulcer? This can only be done by the discovery of other facts which form a part of the history of ulcer; that is to say, the presence of occult blood in the stomach contents or the stools, the occurrence of pain and tenderness which are localized, the radiation of the pain toward the left side of the spine and a characteristic stain with the string test. When these facts are added to those of Reichmann's syndrome, it is proper to classify the case as one of gastric ulcer, and its cure will probably follow treatment for that disease.

It is said that some cases of gastrosuccorrhea are not to be successfully managed without recourse to a gastroenterostomy or to some other drainage operation. I am not prepared to deny the correctness of this statement, but can say that, exclusive of those in which there was food stagnation, I have not encountered such cases. Lavage before breakfast is essential in the treatment of the condition. Large doses of bismuth should be given in conjunction with antacids at an hour that just anticipates the

pain and distress. The hypodermic injection of atropin, once or twice a day, is generally useful. In the beginning the dose should be small, about  $\frac{1}{200}$  of a grain, and should be gradually increased to  $\frac{1}{100}$  or even  $\frac{1}{50}$  of a grain. Also the morning administration of a saline purge following lavage is useful, first in promoting drainage in the stomach and second, in relieving the constipation which is almost always present.

#### PERFORATING ULCER

When the eroding process penetrates the walls of the stomach without producing a limiting perigastritis, there occurs one of the most dramatic and fatal complications of gastric ulcer; that is, perforation with resulting peritonitis. An ulcer may be said to have perforated when it has penetrated the serous coat of the stomach, even though the gastric contents is prevented from escaping into the peritoneal cavity; in other words, when the perforation is slow so that there is time for the formation of adhesions. This benign and strictly localized inflammation may bind the stomach to adjacent parts and so limit the eroding process. In other cases the ulceration goes on burrowing deeply between the viscera, or by eating its way into other organs, it may give rise to a fresh train of symptoms and may mislead the diagnostician as to the real state of affairs. A rather frequent event, when this form of perforation occurs, is the formation of a subphrenic abscess; or, not halting at the diaphragm, the erosion may advance into the thorax and invade the pleural cavities, the mediastinum or the pericardial sac. The abdominal viscera are frequently involved in the slowly burrowing action of these deep-seated gastric ulcers. Acute perforation is first to be considered. very recent ulcer perforation may take place so sud-

denly that there is not time for the intervention of an occluding peritonitis, or, as has been said, after a long period of localized peritonitis, the adhesions may suddenly rupture, thus permitting the escape of stomach contents into the cavity of the abdomen. In either instance this may happen without any unusual physical effort that puts the tissues under strain. Most cases occur when the intra-abdominal pressure is suddenly raised by some unusual exertion, voluntary or involuntary. Hence, the rupture is apt to follow lifting, reaching, coughing, sneezing or straining at stool. Ewald reports a case which appeared in a young woman while dancing. It is more apt to occur when the stomach is relatively full so that distention of the stomach must be considered as a predisposing factor. An ulcer of the pyloric region rarely goes on to perforation, which event more naturally belongs to ulcer of the anterior wall, the lesser or greater curvature, or of the duodenum. Perforation is supposed to occur more frequently when the ulcer is in the anterior wall because this part is more liable to the stress of pressure or blows, but examination of cases lends merely moderate support to this view. Occurring in the posterior wall the perforation is more often limited by adhesions.

Symptoms.—Perforation announces itself by the appearance of intense pain in the upper part of the abdomen. This is usually so agonizing that it overshadows all other symptoms. It is as if a person had been pierced by some sharp instrument. The patient may fall into immediate collapse. Abdominal shock is manifest by the pinched expression of countenance, the pallor, the dilated pupils and the feeble pulse. The abdomen is flat, tense and extremely tender. The blood pressure is raised and soon there will be found an increase in the white blood count.

After a short time the pain becomes less atrocious, the shock decreases and the patient appears to be better. at this time an anodyne be given, the real nature of the situation is so far obscured that the patient may be allowed to develop a general peritonitis, thus greatly imperiling life. The deceitful calm that takes place with lessening pain is looked upon as a sign of improvement, whereas it should be regarded merely as a natural event following a severe and sudden insult to the peritoneum. After the immediate symptoms are alleviated the patient may walk a considerable distance or even attempt to resume work. Very soon, however, the pain returns, although with less severity, a board-like rigidity spreads over the upper abdomen; there is slight febrile reaction with increased arterial tension and heightening of the blood pressure, and the leucocyte count is increased. disappearance of hepatic dullness is improperly regarded as an all-important sign. Although not to be disregarded, it in fact affords but moderate assistance in early diagnosis; first, because it is sometimes inconspicuous, and second, because a distended colon overlapping the liver may give rise to the same sign. Upon careful inquiry it is usually possible to obtain the history that suggests a previous ulcer. It is only in rare cases, or in very dull or unimpressionable people, that the history of ulcer cannot be obtained.

Vomiting may occur, although usually absent, as pointed out by Traube. Mathieu quotes Tuffier as reporting the presence of vomiting in 15 out of 38 cases of perforation. This ratio is far higher than that generally accepted, but the point to be made is that the presence of vomiting does not necessarily exclude perforation of the stomach. In rare cases symptoms are conspicuously absent until the development of secondary peritonitis. Re-

covery is rare except after operation within the first twelve hours.

General Peritonitis.—The later events of perforating ulcer are infrequently seen in these days of prompt abdominal surgery. At times death follows so quickly that there remains little opportunity for study of the later When from misinterpretation of the manifestations. symptoms the attempted rescue by operation is omitted. we have to witness the hopeless picture of a general septic peritonitis. The tale is briefly told. There is the rigid, relatively distended, intensely tender abdomen; there is more or less obliteration of hepatic and splenic dullness; there are perhaps at first a few hurried evacuations of the bowels and then comes obstipation: the bladder acts spasmodically, and then ensues urinary retention. The pain is intolerable and aggravated by the slightest motion. There is vomiting of green fluid, a sighing or Cheyne-Stokes respiration, a hippocratic countenance. There is an exceedingly small, wiry pulse and a rise in blood pressure, but this falls just before the fatal end. The only possible hope lies in the giving of large doses of morphin and in surgical drainage of the abdomen.

Treatment of Perforation of the Stomach.—The therapeutics of acute perforating ulcer of the stomach may be told in these few words. Operate immediately after the rally from the first shock, within the first few hours.

Differential Diagnosis.—There are certain conditions which may be mistaken for perforation of the stomach. For instance, perforation may have occurred in some other part of the alimentary canal or in the appendix or gall-bladder. It is usually possible to exclude the appendix and gall-bladder because of the series of symptoms that precede perforation in these parts. The symptoms of duodenal are nearly identical with those of

gastric perforation. From the foregoing history of the case I have seen the differentiation made, but this does not depend upon the expression of the perforation itself. Embolism of a mesenteric artery or internal strangulation are sometimes excluded with difficulty when the clinical history fails to illumine the situation. Hepatic colic in rare cases comes on so suddenly and is attended with such pain and shock that the question of gastric perforation might be raised; the absence of history of preceding gall-bladder disease and especially the absence of tenderness and induration under the right costal margin are the chief reasons for excluding the trouble.

ACUTE HEMORRHAGIC PANCREATITIS.—Hemorrhage into the pancreas and rupture of a pancreatic cyst are sometimes mistaken for perforating ulcer. In the suddenness of the onset, the intense pain in the upper part of the abdomen, the vomiting and the shock, these conditions bear resemblance to perforative ulcer. However, the pain in pancreatitis is more diffuse. There is an area of resistance and tenderness to pressure, extending transversely across the abdomen just above the umbilicus and describing the region of the pancreas. Though the evidences of abdominal shock are apparent, they are not associated with the symptoms of peritonitis which are present in perforative ulcer. Also in pancreatitis there are often present symptoms of accompanying cholecystitis, or of a preceding gall-stone attack. The rising blood pressure characteristic of perforation I have not seen present in pancreatitis. The blood picture is inconstant in pancreatitis, whereas there is to be expected a rising leucocytosis in perforation. It is only during the onset of pancreatitis, at the moment when hemorrhage occurs that the disease is likely to be mistaken for a perforation. Very soon the somewhat characteristic feature of the disease

would lead one to decide that he was not dealing with Pancreatitis rarely occurs before middle perforation. life; it usually appears in fat people and even in the beginning there is seen a peculiar condition that suggests intoxication. Essentially it is pancreatic hemorrhage that produces the abdominal shock which suggests perfora-This sometimes proves rapidly fatal before hemorrhagic necrosis has had time to develop. I have studied and reported a considerable number of cases of acute pancreatic disease and only in one of these could there have been any difficulty in disproving perforation. This case was that of an elderly woman who had been previously well. She was abruptly seized with agonizing pain in the epigastrium and showed the typical picture of abdominal shock. A physician was called who administered \( \frac{1}{6} \) of a grain of morphin hypodermically. time the woman was in a state of collapse and upon my arrival shortly afterwards she was dving. A very painstaking autopsy discovered no other cause of death than a rather small hemorrhage into the substance of the pan-This case might have well been mistaken for one creas. of perforation.

RETROPERITONEAL, OR TREITZ HERNIA. This may be accompanied by symptoms which, owing to their intensity and abrupt onset closely resemble those of perforative ulcer.

Torsion of the Ureter.—Reference has been made to the close resemblance that sometimes exists between the symptoms of renal lithiasis and those of gastric ulcer. It may also bear repeating that in the syndrome known as Dietl's crisis the urgency of the symptoms is such that the condition might be mistaken for perforation.

Limited Perforation.—This term is employed to describe those cases in which there is perforation without

producing abdominal shock or generalized peritonitis, and has reference to a considerable group. This accident most commonly occurs on the posterior wall of the stomach, but may occur at any point. Although the walls of the stomach are perforated by the ulcer, the stomach contents is not discharged into the abdominal cavity because of the formation of protective inflammatory adhesions. These sometimes develop rapidly into a thick layer, so as to form a considerable mass which can be palpated. Commonly the stomach becomes adherent to some contiguous organ. Between the stomach and the liver, the pancreas or some other part an abscess may develop, and this through infection may extend until it presents for consideration a new problem which seems to have little relation to the stomach; in fact, the communication between the abscess and the stomach may become closed and the abscess continue to enlarge. Under these circumstances the patients present two separate groups of symptoms, viz., those belonging to the ulcer itself, and those belonging to the abscess. Owing to the anatomical arrangement of the upper part of the abdominal cavity and the relation of the stomach to the surrounding parts, the abscess forms in a region depending largely upon the point in which the perforation occurs. Mathieu in his admirable work describes four distinct recesses, in any one of which the abscess may be found: (1) between the diaphragm and the upper surface of the liver: (2) under the liver or between the lower surface of the liver and the anterior surface of the stomach possibly communicating with the preceding; (3) between the posterior surface of the stomach, which forms the front of the abscess, and the duodenum and pancreas which form its posterior wall, with the liver above and the transverse colon below: in other words, the abscess occupies the lesser omental

cavity; (4) in the subduodenal and retroperitoneal space, from which the pus may find its way to the region of the kidney or may follow downwards to the pelvis. Abscesses of this kind may result from perforation of the stomach arising from causes other than peptic ulcer. have met with the condition as the consequence of tubercular ulcer and of slow ulceration occasioned by foreign bodies. One case followed the transit through the stomach wall of a sharp spicule of bone which gave rise to an abscess that was located in the subphrenic and gastrohepatic space. In one instance the abscess lay between the liver and diaphragm and ulcerated thence into the right pleural cavity. The symptoms were those of a diaphragmatic pleurisy and there was a collection of inflammatory exudate, serous in character. After the removal of this it became evident that there was also trouble below the diaphragm. Upon exploration there was found to be an abscess walled off in the pleural cavity that had not been reached by aspiration, and also a large subphrenic abscess. This is a not unusual history with an abscess located between the liver and the diaphragm. another case in which the abscess extended behind the duodenum and was retroperitoneal, it found its way downward and ultimately emptied itself into the colon at the sigmoid. When there remains a free communication between the stomach and the abscess, gas may enter the abscess cavity, giving rise to curious physical signs resembling those of hydropneumothorax. When this occurs in an abscess communicating through the diaphragm with the pleural cavity, it presents a very confusing problem in physical diagnosis. A perforating ulcer may invade the liver or the pancreas and through the resulting inflammation and scar formation it may obstruct the pancreatic or biliary ducts. Reaching upwards it may open into the

pericardial sac as occurred in one of my cases, or it may enter the mediastinum. Osler reports an extraordinary case in which a perforating gastric ulcer extended through the pericardium and entered the left ventricle of the heart.

Occasionally when a perforating gastric ulcer communicates with the colon there may ensue a permanent fistula connecting the two organs. In such a case the stomach is likely to empty itself rapidly, giving rise to diarrhea and the passage of undigested food, and the patient suffers from innutrition.

Reference has been made on page 292 to perigastritis and to the special symptoms to which this condition gives rise. After the healing of chronic ulcer and when its immediate symptoms have subsided, we may be called upon to deal with the special problems relating to the hourglass stomach. This may develop even without perigastritis or adhesions. It is stated that the condition may be produced from the long wearing of very tight corsets in those who have the vertical, or so-called "fish-hook" stomach, without relation to ulcer. In these individuals the greater curvature of the stomach follows an almost perpendicular line down the left side of the abdomen to a point several centimeters below the umbilicus and may reach even to the brim of the pelvis. At this point it bends abruptly upward and to the right where the antrum pylori expands into a pouch of considerable proportions. It is in cases like these that the baneful effects of tightcorset wearing are most striking. The motor function of the stomach meets in these cases with unusual embarrassment in the process of digestion. Patients who have a vertical stomach and who develop gastric atony often suffer from the symptoms of motor insufficiency. We find these symptoms aggravated in those in whom the lower pouch of the stomach, the antrum pylori, becomes distended until something like an hour-glass deformity is developed.

As a complication of gastric ulcer, hour-glass contraction results from the formation of extensive adhesions usually to the pancreas or to the liver. These subsequently contract and hold a segment of the stomach fixed in an elevated position, somewhat as a curtain is looped up by a cord. At times a large ulcer in the process of healing forms an extensive contracting scar which may lead to so much narrowing that an hour-glass stomach is produced, even without the intervention of adhesions. This may occur at any zone of the stomach and therefore we may have a bilocular stomach in which the proximate cavity is small, the partition being formed at a point not far from the cardia, so that the distal pouch is comparatively large; at other times the division is so situated that the two pouches are of about equal capacity, or it may happen that the antrum pylori connects with other parts of the stomach only by a small communicating passage. Occasionally we meet with gastrectasis in the first pouch and not in the second.

Diagnosis.—As stated in a preceding chapter, one of the most characteristic signs of this deformity is the finding in a given case of stomach contents having striking differences in character. The patient may vomit material showing little digestion and having but moderate acidity and, a few minutes thereafter, may eject material in which the digestion is more advanced and in which the gross appearance as well as chemical properties are very different. I have succeeded several times in passing the stomach tube into the second pouch of a bilocular stomach after having previously washed out the first pouch. It is by the use of the fluoroscope or radiograms that we can form the most correct idea as to the extent and the loca-

tion of the hour-glass deformity; and for studying the resulting interference with the motor function of the stomach, this method so far surpasses our other means that the latter have been largely abandoned.

Treatment.—The symptoms occasioned by the hourglass stomach are for the most part those of motor insufficiency and when hyperchlorhydria co-exists they become especially prominent. In some cases we find relative atony in the walls of the proximal pouch but active motion in the distal pouch. When the first pouch is dilated, relief may be had from systematic lavage. In case of hyperacidity, antacids must be given and an unstimulating liquid or semi-liquid diet directed. After the use of these soothing measures the motor symptoms may subside. The improvement is the result of the cessation of spasm in the wall of the second pouch or in that portion of the wall which goes to form the isthmus between the two Should improvement not ensue, an anastomosis or gastroplasty may be advised in order to provide a freer channel of communication between the two pouches. other times it is necessary to make an anastomosis between the proximal pouch of the stomach and the jejunum. and in still other cases it is best to excise the second pouch.

#### PYLORIC STENOSIS AND OBSTRUCTION

Gastric ulcer is often responsible for obstruction at the pylorus, although of course the condition may arise from causes quite apart from gastric ulcer. In rare cases of gastroptosis there is produced a sufficiently sharp angle in the duodenum to retard seriously the movement of gastric contents. Perigastritis from any cause, when located at the pylorus or around the upper part of the duodenum, may, through interference with motion, produce

symptoms that point to a more complete obstruction than actually exists. A familiar instance is the result of pericholecystitis. In case of gastric ulcer in this region, the resulting inflammation around the pylorus may give rise to the formation of dense masses of connective tissue. extensive are these that they may be palpated through the abdominal wall and are highly suggestive of neoplasm. The resulting functional disturbances vary with the location of these inflammatory masses, and with the direct occluding effect which they exercise. More often following gastric ulcer there is marked hypertrophy of the mucosa with induration and thickening of the other walls of the pylorus resulting in marked stenosis. This encroachment may take place on one aspect of the pylorus, the opposite side being free and in appearance quite In other cases the pylorus is uniformly thickened and indurated so that there is only a narrow passage scarcely admitting a lead pencil or even a straw. induration about the pylorus is the result of the gastritis of peptic ulcer hitherto described.

Stenosing Gastritis.—There are a few cases of stenosing gastritis affecting the pylorus, the origin of which is uncertain. Boas was first to describe the condition. It is not easy to differentiate between it and chronic ulcer at the pylorus. The symptoms are those of irritability after taking food, motor insufficiency and ultimately food stagnation and vomiting. The condition produces less pain than attends ulcer and there is absence of hemorrhage. Gastrectasis supervenes in proportion to the duration of the disease and the resisting power of the patient. This stenosing gastritis may be recovered from after a long course of treatment including restrictions in diet.

In the study of obstruction at the pylorus, to these

various benign forms is to be added the consideration of obstruction from malignant disease. Cancer supervenes upon chronic ulcer of the stomach more often than we formerly supposed.

Cancer Following Ulcer of the Stomach.—Cancer as a primary affection attacks the pylorus and, occasionally, but very rarely, the duodenum. The clinical picture of pyloric obstruction following cancer differs from that following ulcer, and the transformation of the ulcer into cancer is accompanied by well-known modifications the symptomatology of which are considered in the article on cancer. (Page 414.)

Diaphragmatic Hernia.—One of the most unusual causes of gastric obstruction results from diaphragmatic hernia. I have met with but one instance of this disease which brought about complete obstruction of the stomach. The patient was a man of 28 years who without apparent cause was seized with violent pain in the epigastrium and thorax. After a few hours, he experienced increasing difficulty in breathing and the respiratory movements greatly increased the pain. There was a sensation of suffocation, with tumultuous action of the heart, and cyanosis and incessant vomiting of somewhat bloodstreaked mucus. Such was the patient's condition when first examined. Percussion then showed over the left chest areas of decreased resonance; breathing sounds were absent in the lower part of the left thorax, but there was heard over this area a most extraordinary assembly of noises, consisting of gurglings and splashings, and sounds of a musical and metallic quality that suggested a hydropneumothorax. This feature of the case had scarcely arrested attention before it was noticed that the most important symptoms were gastro-intestinal. Besides the oft-repeated and violent retching there was marked distension of the abdomen with evidences of violent peristalsis. A stomach tube was introduced, but its passage was opposed apparently about at the cardia and the attempt to wash out the stomach proved a failure. A diagnosis of diaphragmatic hernia, with the protrusion in part both of the stomach and colon into the left pleural cavity was not difficult. A surgeon, Dr. Herman Mynter, was immediately consulted, but the man's condition rendered operation inadvisable. Death ensued quickly and when the autopsy was performed, it substantiated the diagnosis.

No matter what the source of the obstruction at the pylorus, the element of spasm magnifies the stoppage and adds to the difficulty of the passage of gastric contents through the pyloric opening. There are cases in which spasm of the pylorus temporarily obstructs the passage of chyme even without the presence of known structural disease. (See Pylorospasm, page 176.)

Symptoms of Pyloric Obstruction.—Whenever there is continued interruption to the passage of gastric contents through the pylorus, definite symptoms are produced. The symptoms of transient or spasmodic obstruction may be conspicuous, from those of actual stenosis differing only in the fact that they do not persist. They are described in the chapter on dyspepsia.

When there is organic obstruction of the pylorus the symptoms are more or less continuous, they may be further excited by a temporary spasm often caused by a large or irritating meal. However, even with a restricted diet, or during fasting there are ascertainable evidences of delay at the pylorus.

In partial obstruction there are the symptoms of ischochymia or motor insufficiency. The stomach does not empty itself within seven hours after a full meal. Often

there will be found remnants of the meal of the previous evening present in the stomach before breakfast in the morning.

It is the picture of gastrectasis which, indeed, invariably develops provided the case is sufficiently protracted. Anorexia may be constant or, in the case of hyperchlorhydria, the desire for food returns as soon as the stomach is thoroughly cleared out whether by vomiting or lavage. The continued irritation of the gastric mucosa as a result of the presence of the stagnating contents often excites a gastritis which may be so severe as to be very annoying. In the case of an open ulcer, pain is added to the symptom-complex, the suffering increasing in proportion to the distension of the stomach and to the acidity of contents.

When the stenosis is advanced, there are signs of excessive gastric effort in the difficult attempt of the stomach to evacuate its contents. The strong peristaltic waves may be visible through the abdominal wall, and when the accumulation becomes intolerable, vomiting, often of highly acid and perhaps fermenting contents, occurs. When the obstruction is only moderate with little pylorospasm, the symptoms may be merely those of a very irritable stomach, which is sometimes aggravated, sometimes relieved, by the taking of food. It is in these cases that gastrosuccorrhea is found. Sometimes there exists a characteristic Reichmann's syndrome, and yet after considerable distress following a repast, the stomach will empty itself almost normally with perhaps moderate delay. Almost always there is ischochymia and the patient experiences an unnatural sense of contraction in the stomach. When he is exposed upon the table for examination, and when the voluntary muscles are relaxed, it is possible to see transmitted through the abdominal wall the regular rhythmic movements occasioned by the violent gastric peristalsis.

Under these circumstances a fluoroscopic examination of the stomach is most interesting. The shadow produced by the bismuth blended with the food shows that the stomach contents is forced violently into the antrum pylori and against the closed pylorus and is then shot backward and upwards into the fundus of the stomach to return and repeat the performance. The radiogram shows the persistence of the shadow in the stomach long after it would have disappeared under ordinary conditions. When the obstruction is not complete, a small amount of contents may be seen to have escaped into the duodenum. The X-ray offers us a means of estimating the motor power of the stomach and the degree of pyloric obstruction.

Treatment of Pyloric Stenosis.—Since the obstruction may depend partly upon spasm or upon spasm with added temporary hyperemia and edema, the extent of the actual structural stenosis cannot be fairly estimated until after a period of treatment which will allow time for the swelling to subside and the spasm to disappear. This statement deserves the strongest emphasis. When we are dealing with malignant disease or with chronic ulcer it is another matter; but even in chronic ulcer it is wise not to be too precipitate in deciding that artificial drainage is necessarv for the relief of the case. Even granting that operative relief must be practiced before the patient can look forward to a comfortable life, it is generally a wiser plan first to treat the stomach so that it will reach a state of comparative calm, that is, until gastritis has subsided and until the motor excitability has quieted down. I am convinced that cases that have resulted unfavorably with operation might have behaved very differently had this precaution been observed. In neglected cases, in which there has been protracted vomiting with many days of inanition and nights of sleeplessness, the patient is in a pitiable state, quite unfit to undergo a major operation. Under such conditions the surgeon is likely to make the quickest possible anastomosis, fearing that the patient might not survive a pyloroplasty or a Finney's operation which under other circumstances he would regard as the operations of choice. It is true that medical treatment sometimes fails to produce the expected temporary benefit; but a few days' treatment will show whether the plan is advisable or not. It will usually be found that a neglected case which does not undergo temporary improvement by skilful treatment of the stomach is likely to prove equally rebellious to surgical measures. The advisability of treatment before operation is illustrated in the history of the following case.

Case No. XX.—A middle-aged woman had suffered from chronic ulcer for several years, having had repeated hematemesis, and the continued results of pyloric stenosis. She was in a state of alarming prostration occasioned by hemorrhage, starvation and toxemia.

Surgeons wisely declined to operate until her condition was improved. After four weeks of medical treatment there was remarkable gain in the ability to eat, in the state of the blood, in general strength, besides a moderate gain in weight.

The operation (Finney's) was now successfully performed and was followed by the happiest results. Had this case been operated on earlier the patient doubtless would have succumbed.

It must not be too quickly concluded that eventual operation for drainage is imperative. I have had cases in which neglected pyloric ulcer has given rise to obstruction,

to advanced dilatation of the stomach and to dangerous secondary results from inanition and toxemia, and yet have seen these cases recover, not only from the gastrectasis and the food stagnation, but from all evidences of obstruction as well. More often there are occasional recurrences of pyloric irritability that quickly subside after a short course of treatment.

It is proper to introduce here a statement of the obverse side of the picture which may serve as a warning. I refer to cases of neglected pyloric stenosis similar to that just described, in which the prolonged starvation and auto-intoxication have dangerously lowered the patient's resistance, but in which it is impossible to procure a favorable reaction to treatment. As before stated, these cases are unfavorable for operation. The truth to which we should be awake is that pyloric stenosis is a serious condition which with half measures of treatment becomes dangerous. Every consultant who sees a large number of patients suffering from digestive diseases encounters not a few who have received treatment just sufficient to palliate symptoms yet not enough to overcome the trouble. Under such treatment which permits starvation, toxemia and loss of rest, the patient falls into a pitiable condition. Though it may still be possible to lift the sufferer from this grave condition, he ought never to have been allowed to fall into it. Death may occur not only from the direct effects of the grave depression, but from tuberculosis or other intercurrent affections. The following are the methods that have been found most successful in the treatment of pyloric stenosis. first place we must attempt to relieve the spasm and the inflammatory edema. For this the stomach needs complete rest, and this includes rest for the patient as well as for his stomach; that is, the invalid must be put to bed

for a few days and must fast for 24 hours or longer. Meantime rectal alimentation should be practiced and, especially, normal saline solution introduced either by enteroclysis or hypodermoclysis. The need of this course is apparent when it is recalled that a large amount of fluid is discharged into the stomach from the mucosa and subsequently is vomited while absorption from the stomach goes on but tardily so that the tissues are robbed of water. Although the patient is fasting, the stomach should be irrigated once or twice daily with a large amount of warm alkaline solution; and following each lavage there should be introduced through the stomach tube an ounce or two of the milk of bismuth or of bismuth mixture. In addition to these measures hot compresses should be applied continuously over the epigastrium. Ice compresses suit some cases better. Thus may the spasm be relieved and after these measures have been followed for a few days, the stomach will probably attain a state of calm sufficient to allow the commencement of feeding. We should begin with small quantities of milk, expressed beef juice or thoroughly boiled gruel. more than 30 c.c. should be given at one time. The quantity should be so small that it will not induce contraction and over-tonicity of the stomach. Should the stomach prove resentful it should at once be cleared by lavage and the period of rest prolonged a little further. If there is toleration, as is probable, the feedings may be repeated every 2 to 4 hours, and the amount of food gradually increased; but always we should be watchful lest we excite gastric tension, and we should be prepared promptly to empty the stomach when it shows irritability, and to allow it to remain at rest for a few hours before resuming feeding. Slowly the gastric tolerance will increase and for this reason we may gradually widen the dietary. In

addition to milk, gruel and beef juice, we may add purées, the white of egg, meat powder, raw scraped meat, and finally soft toast, thoroughly boiled cereals with cream and sugar, very soft eggs, boiled rice and unsalted butter. If all goes well we may add such solid foods as agree best with the patient, always insisting upon most thorough mastication. The stomach should never be allowed to become distended with food or drink, and lavage should be practiced each night, thus allowing an opportunity for rest of the pylorus until morning. Certain modifications of this plan are sometimes advisable. For instance it may be found well to empty the stomach in the morning and then practice lavage, introducing through the stomach tube a few grams of powdered meat mixed with water or a portion of malted milk. Some patients are made comfortable by the taking of small quantities of a fine quality of olive oil; at times a thin and highly refined vaselin, or white Russian oil may be substituted with advantage. Often benefit follows the taking early in the morning, after the stomach has been at rest during the night, of 200 c.c. or 300 c.c. of warm Carlsbad water, or diluted Abilena water sufficient in amount to produce a gentle evacuation. As soon as possible the patient should be gotten into the open air and there in the lying position allowed to spend much of the day. A short drive on a smooth road, or a half hour ride in an automobile may prove advantageous. The cold spinal douche during the forenoon followed by superficial, stimulating massage, are most serviceable measures. Nerve pressure with vibration over the nerve roots of the upper dorsal vertebrae or the application here of the alternating hot and cold douche, appear to improve the gastric innervation and to lessen the motor insufficiency. It is a mistake to omit these methods of general treatment even

when their adoption requires considerable sacrifice on the part of the patient. They often serve to bring success from the local treatment and from the dietetic measures, where otherwise disappointment is inevitable.

DILATATION OF THE PYLORUS.—Dr. Einhorn recommends in simple pyloric stenosis—that is, in stenosis not associated with active ulcer, erosion or a cancerous infiltration—the stretching of the pylorus by means of his pyloric dilator. This apparatus consists of a duodenal tube attached to the end of which is a distensible, fusiform rubber bag of the proper size. The collapsed bag, attached to the tube, is swallowed, and after having passed the pyloric ring, the bag is inflated; then it is drawn upwards and is made to traverse the pylorus thus stretching the pyloric ring.

Aaron of Detroit reports having had excellent result from the use of the apparatus, and Einhorn reports several successful cases.

SURGICAL TREATMENT.—When we are satisfied that we have accomplished the most possible by the treatment herein described and find that there yet remains so much stenosis that full nutrition is impracticable; when we find that a meal only sufficient to sustain the patient brings about gastric tension and when it is evident that the balance of nutrition cannot be maintained within the limitations of feeding made necessary by the symptoms in the case; in short, when we find that the patient is not cured of his symptoms by medical measures, it is our duty to urge artificial drainage of the stomach; and we should see to it that the operation is done by a man who is thoroughly familiar with and skilful in the surgery of the stomach. It has been my experience to see a number of cases of pyloric stenosis worse after operation than before. Most surgeons have come to select gastrojejunostomy as the operation of preference, and indeed it is the only feasible operation in certain cases. However, I predict that the frequency of this operation will undergo a great decline. Contrary to the experience of some I have seen only the most favorable results from pyloroplasty and from Finney's operation when performed by thoroughly skilful men.

### CHAPTER XIII

#### GASTRIC EROSIONS

In 1894, Einhorn described a condition characterized by continued dyspeptic symptoms with occasional exacerbations, accompanied by weakness and anemia. special feature was the presence of small masses of gastric mucous membrane removed with the wash water on the practice of lavage in a fasting stomach. The nature of the tissue was shown by histological examination. fragments were flesh-colored, sometimes red, and were evidently freshly separated from the mucous membrane of the stomach. Einhorn recognizes the condition as a definite symptom-complex, which he calls "gastric erosion." For several years Dr. Allan Jones and I had observed these cases without being able to come to a decision as to their precise significance. At first I was inclined to agree with Elsner in attributing the source of these fragments of gastric mucosa to a chronic gastritis. It must be confessed, however, that in most cases there is an absence of mucus such as is ordinarily seen in gastritis and, on the whole, the facts set forth by Einhorn cannot be successfully disputed.

Post-mortem studies of the condition have been lacking. Recently, autopsy findings in two cases, in which the clinical history corresponded closely to the description given by Einhorn, have been reported by N. W. Jones of Oregon. He speaks of the disease as a variety of hemorrhagic erosion, although distinct from the type of Dieulafoy. He apparently bases his conclusions upon the post-

mortem appearance of the mucosa, not upon the clinical history. The erosions found in these cases were multiple and pigmented, or blood-stained. From the description given one could not distinguish between them and other types of hemorrhagic erosions, such as are described by Hayem. (See Peptic Ulcer, page 239.) The gastric mucosa showed evidence of gastritis with round cell infiltration but with absence of connective tissue proliferation. N. W. Jones would classify the disease as a special form of gastritis; not ordinary gastritis as held by Elsner, not a non-inflammatory state as believed by Einhorn. The specimens warrant the conclusion that there were hemorrhagic erosions without clinical hemorrhage occurring in cases of chronic gastritis. More than this is not proved by these cases.

As yet one is scarcely justified in classing the erosion of Einhorn as a special disease, although eventually such may turn out to be the case. It has been held by some that the trauma attending the use of the stomach tube, the tissues having an unusual degree of friability resulting from some form of gastritis, is responsible for the dislodgement of these fragments of the mucosa, but of this I am not convinced. I have observed slight oozing of blood in cases of gastric erosion when vomiting occurred while the tube was in the stomach. Einhorn relates a like experience. This at least indicates a vulnerable mucous membrane, if not gastritis.

For years my attention has been attracted to the coincidence of pyorrhea alveolaris with gastric erosion. The most intractable case of erosion which I have encountered was accompanied by an equally rebellious pyorrhea. Recently bacteriological study has been made of the pus coming from one of the pockets under the margin of the gum. There have been found present a variety of organisms,

as follows: staphylococci, occasional diplococci, long bacilli, besides an organism that appeared to be the streptococcus mucosus capsulatus. A special form of spirochete has been described which has been found only in the pus of pyorrhea. The stomach contents in one case showed a total acidity of 39, combined chlorids, 21; free hydrochloric acid, 15; lactic acid was absent; there was



Fig. 25.—Fragments of Tissue Removed from Wash Water in Case of Gastric Erosion.

no unusual amount of mucus, although occult blood was present. This describes the stomach contents typical of the condition. The intestinal digestion was satisfactory, and there was no occult blood.

The fragments of tissue recovered from the wash water in these cases vary in size from that of a mere particle to pieces 1 or 2 cm. in length. In order to convey an idea on this matter there is shown in the accompanying figure a representation of the tissue obtained at one lavage showing the actual number and size of these fragments. (Fig. 25.) The histological study of these was made by Dr. H. U. Williams. Photomicrographs of the sections are here reproduced. (Fig. 26, Fig. 27.) Dr. Williams concludes: "The sections show a moderate increase in the loose lymphoid tissue between the glands. Occasionally the gland tubes are somewhat dilated



Fig. 26.—EINHORN'S GASTRIC EROSION. Section by H. U. Williams, from one of the fragments shown in Fig. 25, coming from the case of gastric erosion referred to. There is moderate increase in loose lymphoid tissue between glands. A few gland tubes are very dilated, some slightly cystic.

and even slightly cystic. There is one small spot of brown stain (not pigment) in the tissue, probably meaning a minute hemorrhage; there is no fresh hemorrhage. There is probably a trifling increase in the amount of interstitial fibrous tissue. Occasionally a few leucocytes may be seen near the surface and in the lumena of glands. On the whole, the amount of pathological change

shown in the specimen is slight." It is evident that the mucosa from which these fragments escaped was not a healthy one and yet there is not conclusive evidence of gastritis. If it were a common practice to examine critically the wash water recovered in the routine practice of lavage undoubtedly the finding of fragments of mucous membrane would not be a rare experience. In this con-

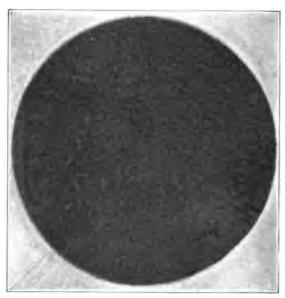


FIG 27.—EINHORN'S GASTRIC EROSION. Shows small, brown stain, probably from minute hemorrhage. No fresh hemorrhage. Trifling increase of interstitial fibrous tissue. A few leucocytes are seen near the surface in lumina of glands.

nection attention is called to the useful contrivance of Dr. Anthony Bassler called a "sieving pail" for lavage water which permits of the convenient collection of all solid matter discharged in the wash water with the lavage. (Fig. 28.) A comparison of the section from my specimen as described by Williams with the reproductions made from cases of gastric erosions by Prof. Hayem <sup>1</sup> (See Peptic

<sup>&</sup>lt;sup>1</sup> Hayem: Arch. des Mal. de l'Appar. Digest., July, 1911.

Ulcer, pages 240, 241, 243) leads to the belief that a distinction is to be made between these lesions. These specimens differ also from those described by the late Prof. Dieulafoy in his text-book of medicine.

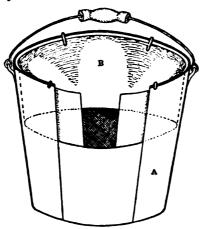


Fig. 28.—Sieving Pail of Bassler for Lavage Water. a, pail; b, sieve; front of both laid open.

Symptomatology.—Patients having gastric erosions complain of the sensation of gastric distress and soreness sometimes amounting to a dull pain, and a feeling of gastric tension. The symptoms are made worse by eating. In the case herein referred to there was complaint of a disagreeable "drawing sensation" under the left costal border. I have also found unusual sensitiveness over the abdominal sympathetic centers and along the course of the abdominal aorta.

Treatment.—Although the condition is sometimes refractory to treatment, it is often promptly improved and apparently cured by local applications of silver solution. Tincture of chlorid of iron is a useful remedy in some cases, bismuth subgallate in others. The diet should be restricted to soft unstimulating, non-irritating foods and all solid food should be very thoroughly masticated.

#### CHAPTER XIV

# CANCER OF THE STOMACH: GENERAL CONSIDERATIONS AND PATHOLOGY

Introduction.—Whatever may be the nature of cancer, it is evidently dependent upon some unknown cause which deranges biologic laws common to all vertebrates and which results in producing "the unceasing proliferation of the cells of the growth, cells which may be undistinguishable from those of normal tissues." (Bashford.)

Aside from the doctrine that cancer originates from the entrance into the tissues of some external agent, the various theories so far offered as explanatory of malignant growths are, it seems to me, incompatible with established principles of evolution, that is the law of "adaption and the survival of the fittest." The connective tissue theory of Virchow; the "cell rests" theory of Cohnheim, and the later theory of Beatson which supposes the transformation of somatic cells into those of reproductive tissues, or any one of the other recognized hypotheses for the explanation of the development of cancer, are faced by the fact that a strange and definite irruption of biologic harmony has not been eliminated in the evolutionary ages by the process of natural selection. It would seem to be inconsistent with what we know of the law of the "survival of the fittest" that a definite destructive disturbance in the biologic processes, common to all vertebrates, should have survived.

Notwithstanding that cancer behaves unlike any known infective disease, the fact remains that in respect to its

wide prevalence in various orders of life its history conforms to that of the infections; furthermore, the progressive increase in cancer, as indicated by statistics is accounted for by attributing its cause to some external agent. In addition there has been thought to be a preponderance of the disease in regions having a special geologic formation; the occurrence especially in certain districts; the tendency to develop in the two sexes in different proportion at one time and place as compared with another, all seem to point to some external cause.

There is no subject in internal medicine more disspiriting to the physician, no disease that produces more despair in the patient than cancer of the viscera; and there is no internal organ so often invaded by this disease as the stomach. It is but natural that the greatest activity has been shown, in attempting not alone to solve the nature of cancer, but also to reach a diagnosis so early that intervention of surgery may be efficacious.

No diagnosis is more easily made than that of gastric cancer after it is well developed, especially when it occurs without complication in a previously healthy person. On the other hand nothing is more difficult than the early diagnosis of cancer of the stomach; that is, the early diagnosis from a surgical point of view, especially if the disease attacks a person whose health has continually been impaired by diseases the symptoms of which mask those of cancer. In many such cases cancer is not suspected until it is far advanced. As is well known, cancer most often develops in middle life, in women at the time of the climacteric, and in men at the age when stiffening arteries lead to a decline in functional power and general activity, without any offsetting decrease in the inveterate over-indulgence in eating, drinking and smoking, but with the consequences of impaired metabo-

In either sex this is a period of life in which the general health usually undergoes changes which the laity have come to recognize and to accept as a matter of course, and the early symptoms of cancer of the stomach are therefore too often attributed by the patient to fanciful or ill-defined causes, which tend to allay suspicion and to satisfy inquiry until the victim eventually recognizes that something is going seriously wrong with him. matter becomes more difficult because in many instances cancer of the stomach follows chronic ulcer or other forms of digestive trouble. Finally, to fill up the measure of embarrassment, we find the disease making itself the companion of some preceding affection, which of itself has produced cachexia. Thus in chronic nephritis, cirrhosis of the liver, prostatic diseases, spinal cord lesions, the chronic infections, diabetes and the other metabolic derangements, we find depreciation of the general health, often with accompanying dyspepsia, which serves to deceive the patient who is harboring cancer.

Though cancer may thus attack those already depressed from other diseases, it usually begins in those previously healthy. This is important in diagnosis; the abrupt onset of symptoms with rapid failure of health without other obvious cause at once suggests the possibility of malignancy. Yet this well-known rule may lead to error in the opposite direction; the temptation to assume must be repressed.

Even in the absence of preconception even when the patient is under expert professional observation, cancer may progress undetected. Of course this is not true in classical cases in which typical manifestations of the disease appear in orderly succession. When there is a sudden decline in appetite and in gastric digestion, when there is a loss of weight, anemia and cachexia, when the

gastric secretion fails, when vomiting develops and when there is hematemesis and palpable tumor, no one could hesitate in the diagnosis of cancer. Too many physicians remember merely the classical, text-book description of the disease as it appears when well advanced. It is more important for us to understand its manifestations in the earliest stage. In this connection one should recall the pregnant remarks of Gaston Lyon, ''La symptomatologie est donc la plus variable et, s'il faut se souvenir des descriptions classiques, il faut aussi savior les oublier parfois, sinon, on s'exposerait à de graves erreurs de diagnostique'—surely, if it is necessary to remember the classical descriptions of the disease, it is also necessary to know how to forget them sometimes, lest we expose ourselves to grave errors in diagnosis.

#### INCIDENCE OF CANCER OF THE STOMACH

The relative frequency of cancer appears greater than it is for the reason that a lethal termination serves to identify it; whereas a proportion of other diseases of the stomach recover without diagnosis. The statistics of hospitals, in which diagnosis is presumably more accurate than in general practice, cannot be taken as a criterion for the reason that a greater proportion of the very ill seek hospital relief; hence relatively more cases of cancer. For instance, in his study of "Post Mortem Diagnosis of the Chief Causes of Death in 3000 Persons," Richard Cabot found 83 cases of carcinoma of the stomach and but 65 cases of ulcer including those in the duodenum. This, of course, is not in accord with the relative frequency of the two diseases.

Writing in 1900 after a survey of available statistics, Osler and McCrae state: "The rather scanty data do

<sup>&</sup>lt;sup>1</sup> "Diagnostique et Traitement des Maladies de l'Estomac."

not justify a positive conclusion as to the increase of cancer of the stomach."

Death from cancer in the United States occurs in practically the same ratio in urban and rural districts. A slight preponderance in cities, is probably owing to methods of diagnosis and to the fact that country dwellers often seek relief at, and ultimately die in, city hospitals. Per 100,000 of population in 1907, 79.6 per cent died in cities and 73.9 per cent in the country.<sup>2</sup>

The apparent increase of cancer in the country is shown by comparison of the number of deaths in 1904 (70.6) and in 1908 (74.3). During this period there was but slight increase of cancer of the mouth (2.1 as compared with 2.5). However, the increase in cancer of the stomach was from 26.4 to 29, and in cancer of the intestines from 10.3 to 11.8. During this period there was an increase in cancer from all causes, per 100,000 population, of 4.3, and an increase of cancer of the stomach and intestines of 4.1. The increase appears to be progressive from year to year. During 1908 there were reported 33,465 deaths from cancer of all forms, and of these 13,044 were from cancer of the stomach, or about 39 per cent.

Although the increased mortality from cancer can in part be attributed to increased longevity, whereby a larger number of people reach the cancer age; although in part it may be explained by more accurate diagnosis, a careful study of the question indicates an advance in the mortality.

There is a matter for reflection in the distribution of the disease. Of 10,803 cases, 5,976 occurred in cities and 4,827 in rural districts. The proportion per 100,000 population is: urban, 80.5; rural, 68. There is but trifling difference between the incidence in city and country in

<sup>&</sup>lt;sup>2</sup> U. S. Census Report.

several states; yet in Maryland the figures are, for the whites in cities, 34.2; in the country, 17.3; among the colored in the cities, 25.1; in the country, 6.3. One cannot but suspect that the reporting of cases among the rural dwellers in Maryland is incomplete, and that this applies especially to colored people.

In Massachusetts the ratio is: urban, 29.1; rural, 35.5. In Maine it is: urban, 33.3; rural, 41.6. The disease is relatively more frequent in the country in Vermont, South Dakota, Rhode Island; in the cities, in California, Colorado, Indiana, Michigan, New Jersey and Pennsylvania. Town and country statistics are nearly alike in New York and Connecticut.

We should not too hastily conclude that these varied results are solely because of irregularities in reporting, for equally remarkable returns, as is shown in statistics which follow, appear in countries where record keeping is highly developed. Evidently we are approaching a time when the statistical study of cancer of the stomach will greatly illuminate the subject.

The following statistics made available through the courtesy of Dr. Gaylord of the New York State Laboratory for the Study of Malignant Diseases (Buffalo) are instructive, not alone as to incidence of cancer of the stomach but also as to its etiology.

## THE LOCALIZATION OF CANCER AS TO THE ORGANS AFFECTED

### IN BAVARIA AND OTHER COUNTRIES

Kolb found in Bavaria, on the basis of accurate statistics prepared by the Bavarian Committee for the Investigation of Cancer, that cancer of the stomach represented 58.7 per cent of all cancer in men and 41.1 per cent of all cancer in women. If to these figures be added all cases

of cancer of the peritoneum and of the organs of digestion it will be found that cancer of the organs of digestion below the diaphragm constitutes 79 per cent by men, and 80.3 per cent by women of all cancer. It will be noted that the proportion is in excess of the American figures.

LOCALIZATION OF CANCER AS TO THE ORGANS AFFECTED (KOLB)

Bavaria	Switzer-	England	Hungary	Italy s	U. S.	Sweden
1.9 4.7	2.3 12.8	9.8 6.3	6.8 2.9	13.0	9.5	9.3 4.6
6.6	15.1	16.1	9.7		=	13.9
65.4 5.7 5.7 0.4	65.5 3.6 2.6 2.4	36.7 7.3 10.2 0.2	65.5 6.7 2.2	71.5	57.6 ? 5.5	61.5 9.4 —
77.2	74.1	54.4	74.4		_	_
2.1	1.4	2.5	0.2		9.2	
0.7 1.3	0.4 2.7	1.2 1.5	0.7 0.4	3.9	1.7	2.2 2.1
2.0	3.1	2.7	1.1		_	4.5
48.8 4.9 2.9 0.9	50.1 3.9 2.1 0.1	28.7 6.8 5.9 0.2	45.4 6.4 1.5 0.1	49.5	37.1 ? 3.5	34.2 7.8
		41.6	53.4	] [		
57.5	56.2	41.0	00.4	11		
57.5 5.1 19.5 8.1	3.1 22.7 10.7	3.6 24.9 16.6	0.5 33.1 6.6	29.4 10.8	7.7 29.3 15.8	21.4 23.9
	1.9 4.7 6.6 65.4 5.7 5.7 0.4 77.2 2.1 0.7 1.3 2.0 48.8 4.9 2.9	1.9 2.3 4.7 12.8 6.6 15.1 65.4 65.5 5.7 2.6 0.4 2.4 77.2 74.1 2.1 1.4 0.7 0.4 1.3 2.7 2.0 3.1 48.8 50.1 4.9 3.9 2.9 2.1	1.9 2.3 9.8 4.7 12.8 6.3 6.6 15.1 16.1 65.4 65.5 36.7 5.7 2.6 10.2 0.4 2.4 0.2 77.2 74.1 54.4 2.1 1.4 2.5  0.7 0.4 1.2 1.3 2.7 1.5 2.0 3.1 2.7 48.8 50.1 28.7 4.9 3.9 6.8 2.9 2.1 5.9	1.9 2.3 9.8 6.8 4.7 12.8 6.3 2.9 6.6 15.1 16.1 9.7 65.4 65.5 36.7 65.5 5.7 3.6 7.3 6.7 5.7 2.6 10.2 2.2 0.4 2.4 0.2 — 77.2 74.1 54.4 74.4 2.1 1.4 2.5 0.2 0.7 0.4 1.2 0.7 1.3 2.7 1.5 0.4 2.0 3.1 2.7 1.1 48.8 50.1 28.7 45.4 4.9 3.9 6.8 6.4 2.9 2.1 5.9 1.5	$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$

<sup>&</sup>lt;sup>3</sup> Larynx and thyroid gland are included in statistics for Italy.

STATISTICS OF VARIOUS AUTHORS BASED UPON AUTOPSIES IN PATHOLOGICAL INSTITUTES

	Munich		Berlin		Klausen- burg
Men.	Rieck	Redlich	Feilch- enfeld	Riechel- mann	Buday 1889-1905
Lips, mouth, tongue, throat Esophagus	5.1 6.5	4.9 17.3	3.6 21.3	1.8 20.0	13.4 6.7
Total	11.6	22.2	24.9	21.8	20.1
Stomach, liver, pancreas Intestines Rectum Other digestive organs	47.3 8.1 9.4 1.6	43.5 4.5 6.7 3.5	44.7 5.5 5.1 1.2	50.0 4.3 5.1 3.9	42.0 6.7 6.7 0.8
Total	66.4	58.2	56.5	63.3	56.2
Peritoneum, mesentery	0.9	_			1.7
Women.					
Lips, mouth, tongue, throat Esophagus	0.8 0.7	1.0 2.8	1.6	1.4	1.4
Total	1.5	3.8	1.6	1.4	1.4
Stomach, liver, pancreas Intestines Rectum Other digestive organs Total	28.7 3.4 5.3 1.2	31.4 7.0 5.6 10.8	28.4 5.0 5.5 9.1 48.5	37.0 4.5 2.3 9.5	22.5 2.0 1.0 3.9
Peritoneum, mesentery	1.9				
Uterus Ovary Vagina, vulva	32.7 4.3 2.4	17.4 5.6 1.4	20.5 4.7 4.4	24.6 4.0 1.4	34.3 22.5 2.9
Total	39.4	24.4	29.6	30.0	59.7
Mamma	11.6	12.2	12.6	8.3	7.0

An examination for the different forms of cancer as shown by countries, indicates to what extent cancer is a

disease of the gastro-intestinal canal. With cancer of the peritoneum included it represents in Bavaria 85.9 per cent of all cancer; in Hungary 84.3 per cent; in Italy 84.5 per cent; in Sweden 84.8 per cent; in Switzerland 90.6 per cent, and the only exception to this astonishingly high proportion is England which shows 73 per cent. That these figures, although much higher than those previously taken, are approximately correct is shown by comparison with the table taken from autopsies in pathological institutes. In Klausenburg, cancer of the gastrointestinal tract and peritoneum totals 78 per cent, in Munich 78.9 per cent, and in Berlin 80.4 and 85.1 per cent. In Bavaria, Switzerland, and Hungary figures for stomach, liver and pancreas together with those of the other abdominal digestive organs, show a corresponding percentage. Cancer of the intestine and rectum shows a much higher percentage in England and a much lower percentage in Switzerland and an intermediate position in Bavaria. Cancer of the digestive tract from the lip to the esophagus is much less frequent as shown by the table in Bavaria than in Hungary, Switzerland and England. Cancer of the lip and mouth is particularly frequent in Hungary and England, while in Sweden this is true of the esophagus.

Frief has made the observation, which Redlich endorses, that cancer in the human race shows a predilection for the organs immediately adjacent to the orifices for the reception of food and air, and whereas these orifices are more exposed to mechanical irritation in men than in women, the percentage is higher for the male than the female. Alcohol seems to be concerned in this irritation, especially the stronger types of spirit, which may account for the lower figures in countries where beer is consumed to the exclusion of stronger drink. The statis-

tics of the pathological institute in Munich show exceptionally few cases of cancer of the esophagus. Boas found that 40 per cent of all cases of cancer of the esophagus treated by him were spirit drinkers. Kolb has also observed the association in certain localities in Switzerland where cancer of the esophagus occurred most frequently in men who were frequent users of strong spirits and tobacco, whereas women of the same district were practically free from the disease. In the region of Wagen, Canton of Berne, he observed that 36 per cent of all carcinoma in men was in the esophagus and represented 425 cases per million of living men which is nearly equal to two-thirds of the entire mortality from cancer in Prussia.

In the Bavarian statistics of 1905, Kolb found that five out of nine cases of cancer of the esophagus were hard drinkers of spirits.

The following table gives the percentage of cancer of the mouth and of the esophagus in three countries Bavaria, Switzerland and England.

	Men	Women
Lips and mouth:		
Bavaria	1.9%	0.7%
Switzerland	2.3	0.4
England	9.8	1.2
Esophagus:		l
Bavaria	4.7	1.3
Switzerland	12.8	2.8
England	6.3	1.5

The consumption of spirits in England is difficult to determine. For 1905 Kolb estimated it at 25 liters per capita and he observes that this is certainly a higher consumption than in Bavaria.

England, when compared with all other countries, pre-

sents certain peculiarities in the distribution of cancer. First of all, the relative rarity of cancer of the stomach, liver and pancreas, and the comparative frequency of cancer of the upper organs of digestion, and of the intestines and rectum. The same holds for women in England, and in them the relative rarity of cancer of the stomach is even more marked. The women of England, as well, however, as the women of Italy, the United States and Sweden, show a high percentage of cancer of the organs of reproduction. The total cancer death rate in England is distinctly less than in Bavaria and Switzerland. Of the living, 742 die of cancer in England, 966 and 1097 respectively in Bavaria and Switzerland.

One possible explanation of the relative rarity of cancer of the stomach in England, as pointed out by Kolb, can be found by dividing all forms of cancer into three classes: accessible, intermediary, and inaccessible.

DISTRIBUTION OF CANCER ACCORDING TO DIAGNOSTIBILITY

		Men			Won	ren	
	Bavaria	Switzer- land	England	Bavaria	Switzer- land	England	Italy
Accessible	7 7.5 85.4	4.7 5.2 90.1	22.4 15.5 62.2	13.1 21.2 65.7	12.9 23.2 63.8	22.9 30.6 46.5	16.1 29.4 54.5

From this table it will be seen that in Bavaria and Switzerland in men the inaccessible and difficult to diagnose types of cancer are from 85 per cent to 90 per cent, whereas in England they are but 62 per cent. In women a similar discrepancy is noted. Kolb refrains from drawing the conclusion that an explanation of this smaller per cent in England may lie in the diagnostic methods of that country.

Unquestionably these statistics must be affected by the average period of life in the various countries. In what may be called the cancer age (that is beyond fifty), only 14 per cent of the men are living in Bavaria, in England 16.2 per cent, in Switzerland 17.7 per cent, which means that one-sixth more are alive at this period in England, and a full quarter more are alive in Switzerland at this age than in Bavaria. A similar relation exists with women.

From the table following showing the relative susceptiblity to cancer at the various age periods, it will be seen that predisposition increases progressively up to the seventieth year and even beyond, which is in accord with the statistics of other countries. For instance, in England from 1891 to 1897 the number of deaths from cancer at the various age periods per million living was:

						25-35 yrs.					
Men	32	18	18	32	49	98	386	1283	3050	4977	5432
Women	26	12	12	25	39	180	918	2328	4045	5555	6135

## LOCATION OF CANCER AMONG CASES IN ENGLAND

Men.	25-35 yrs.	35-45 yrs.	45-55 yrs.	55-65 yrs.	65-75 yrs.	75-85 yrs.	Over 85 yrs.	Total
Esophagus	2	22	125	293	390	358	190	47
	28	154	555	1505	2671	2696	1725	272
	10	32	103	277	542	631	312	55
	12	36	143	404	748	852	637	75
Women.								
Esophagus Stomach, liver, pancreas Intestine Rectum Uterus, ovary, vagina Mamma	5	14	33	57	117	135	105	15
	23	154	574	1464	2505	2636	1775	294
	9	36	128	324	609	698	557	70
	12	38	124	282	467	561	387	61
	71	372	827	1081	1134	1508	2182	255
	21	173	469	720	1002	1443	2123	170

# Comparison of Cancer Cases per Million Living in Sudbayern and Nordbayern

## SUDBAYERN.

Men.	30-40 yrs.	40-50 yrs.	50-60 yrs.	60-70 yrs.	Over 70 yrs.	Total
All cancer cases Stomach, liver, pancreas Intestine Rectum	259 165 10 12	1245 781 102 43	3575 2335 255 270	7650 5020 566 482	11551 6282 675 299	1151 728 81 72
Women.					ŀ	
All cancer cases Stomach, liver, pancreas Intestine and rectum Uterus Mamma	574 145 33 257 37	1946 652 130 710 174	4203 1691 310 1177 416	7632 4027 627 1254 649	9604 5198 1029 878 745	1445 651 119 321 123
	ORDBAY	ERN.				
MEN. All cancer cases	169 132 14	754 589 44 30	2439 1723 91 75	6181 4230 320 270	8198 5217 353 <b>45</b> 8	840 564 39 35
Women.		1	Ì		Ì	ł
All cancer cases Stomach, liver, pancreas Intestine and rectum Uterus Mamma	361 125 27 95 40	1262 562 93 276 138	2951 1595 165 429 213	5886 3495 456 653 290	7824 4453 670 563 480	1002 546 74 134 68

Comparison of age periods in	0-30 yrs.	30-40 yrs.	40-50 yrs.	50-60 yrs.	60-70 yrs.	Over 70 yrs.	Total
Bavaria { men	60.8	12.8	10.2	8.2	5.2	2.8	100.
	59.3	12.9	10.1	8.5	6.0	3.2	100.
	58.6	12.8	11.3	8.9	5.9	2.9	100.4
	56.9	13.0	11.7	9.4	6.4	3.1	100.5
	61.6	13.9	10.6	7.2	4.4	2.4	100.1
	59.9	14.2	10.5	7.4	4.9	3.0	99.9

COMPARISON OF AGE PERIODS AND DIGESTIVE ORGANS AFFECTED IN MEN AND WOMEN

пинапозіть	7.2 6.17 6.25 6.28 6.28 6.28 7.3 7.3 7.3 7.3 7.3 7.3 7.3 7.3 7.3 7.3	0.55 0.57 1.11 1.11 16.4.12 2.12 2.55 2.56 1.58
въэтэп.вЧ	1.9 1.9 1.9 1.9	9 45 48 5 6 8
Gall- Bladder	11111111	14.5 114.5 33.7 115.7 100
Təvi.I	4.0 0.2 0.2 0.0 0.0 0.0 0.0 0.0 0.0 0.0 0	0.6 0.6 0.6 25.6 35.7 20.4 4.2
Кесtum	200 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	11.9 12.0 12.0 12.0 12.0 12.0 12.0 13.0 10.0
Intestine	20 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	0.2 0.2 0.7 0.7 0.7 19.6 36.9 24.6 3.5 3.5 99.9
Stomach TavirI &	0.3 2.1 14.0 31.6 32.3 18.6 1.0	0.3 0.0 0.3 0.0 8.4 8.4 37.9 19.4 19.4 99.9
Stomach	2.7 10.1 25.0 38.3 21.3 2.13	20.0 20.0 3.0 3.0 3.1 3.1 3.1 100
Esophagua & Stomach	25.5 7.5 35.0 32.5 15.0	26.3 26.3 36.3 36.3 36.3 36.3 36.3 36.3
Беорћадив	0.3 10.5 10.5 27.8 35.5 20.4 4.0	4.1 4.1 4.1 4.1 99.9
<b>Ж</b> ои <b>£</b> h	21-22222	22.55   22.55   30.00   32.55   100
ТопдпоТ	12 2 2 2 2 2 1   5	5.9 5.9 29.4 35.3 11.6 11.7 999.9
sqirI	1   2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2	13.3 13.3 26.7 46.7 6.7
Мен.	0-20 yrs. 20-30 " 30-40 " 40-50 " 60-60 " 70-80 "	Women. 10-20 ". 20-30 " 30-40 " 40-50 " 60-60 " 60-70 " 70-80 "

From this it will be seen that the predisposition to cancer at the age of seventy is fifty times greater than it is at the age of thirty, the percentage of living at seventy being, of course, many times smaller. From seventy to seventy-five years of age the susceptibility appears to decrease.

That the predisposition to cancer is not alone dependent upon old age is shown by the fact that cancer of the uterus and ovaries decreases after the period when these organs are no longer active.

CANCER CASES PER MILLION ACCORDING TO AGE PERIODS

Men.	Entire popu- lation	0-15	15-30	30-40	40-50	50-60	60-70	Over 70 years
Oberbayern	1044	17	37	285	1209	3500	7051	10005
Niederbayern	829	27	51	214	841	2467	4800	5104
Schwaben	1351	25	47	204	1315	3904	8667	10862
Oberpfalz	960	10	34	268	886	2687	6340	7231
Mittelfranken	944	24	71	268	967	2965	7088	9592
Pfalz	766	19	38	139	676	2748	6654	9427
Oberfranken	826	18	14	162	718	2464	6002	6830
Unterfranken	935	37	88	216	877	2078	5921	8318
Kingdom	966	57	48	225	975	2906	6607	8493
Women.								
Oberbayern	1385	20	71	561	1858	4433	7477	9045
Niederbayern	1125	13	61	599	1570	2937	5387	5788
Schwaben	1552	16	43	601	2109	3833	7913	10540
Oberpfalz	1016	13	36	406	1367	3187	5066	6063
Mittelfranken	1194	12	53	494	1859	3439	6161	5423
Pfalz	1018	19	60	349	1238	3170	7152	9573
Oberfranken	919	21	23	399	1252	2630	5132	5777
Unterfranken	1108	23	88	341	1297	3018	6004	9912
Kingdom	1193	17	58	481	1616	3597	6435	8263

Kolb's studies of the geographical distribution of cancer for the different sections of Bavaria are of great interest. For the total male population the maximum is found in Schwaben, and the minimum in Pfalz, and the difference is so great that the maximum is seven-fourths

more than the minimum. From maximum to minimum by districts the sequence is as follows: Schwaben, Oberbayern, Mittelfranken, Unterfranken, Oberpfalz, Niederbayern, Oberfranken, and Pfalz. If the distribution is taken by age periods it will be seen by the table on page 392 from Kolb that if the cases under forty are eliminated, Niederbayern and Oberfranken give the most favorable showing, and Unterfranken at all age periods is below the average.

DEATH BY CANCER OF THE STOMACH, LIVER AND PANCREAS, PER MILLION

Men.	0-30 yrs.	30-40 yrs.	40-50 yrs.	50-60 yrs.	60-70 yrs.	Over 70 yrs.	Entire popu- lation
Oberbayern	636	7	190	727	2080	4526	5763
Niederbayern	550	8	94	611	1645	3408	3031
Schwaben	900	8	121	883	2756	5820	7030
Oberpfalz	682	4	182	603	1925	4808	4821
Mittelfranken	562	4	118	540	1583	4612	5424
Pfalz	472	4	46	446	1743	4337	5269
Oberfranken	597		126	493	1851	4532	4624
Unterfranken	567	7	112	585	1373	3758	4858
Kingdom	618	5	129	626	1932	4777	5124
Women.							
Oberbayern	592	3	113	575	1622	3878	4857
Niederbayern	482	2	105	426	1213	2731	3536
Schwaben	760	3	213	794	1809	4270	5754
Oberpfalz	500	6	158	429	1692	2903	2867
Mittelfranken	531	7	107	519	1584	3256	4895
Pfalz	563 .	7	110	658	1773	4153	5645
Oberfranken	486	7	118	593	1393	3005	3063
Unterfranken	581	7	147	429	1586	3672	4580
Kingdom	567	5	130	559	1501	3555	4478

Leaving out all cases under forty years of age, the mortality from cancer of the stomach represents two-thirds of the total deaths from cancer. It is seen that in Nordbayern the percentage of deaths from gastric cancer is below the average (this is also true of the intestine and colon), whereas in Sudbayern cancer of the stomach is

well above the average of the entire country and represents three-fourths of all cancer.

CANCER CASES IN SWITZERLAND PER MILLION LIVING

Men.	0-30 yrs.	30-40 yrs.	40-50 yrs.	50-60 yrs.	60-70 yrs.	Over 70 yrs.	Total
Stomach, liver, pancreas Intestine Rectum	5 4 0.2	125 17 9	696 31 20	2045 91 66	4015 219 160	4573 241 241	652 36 26
Women.							ļ
Stomach, liver, pancreas	5 2 0.1 5	101 16 8 152 42	504 38 23 477 150	1386 103 42 762 308	2750 183 113 781 424	3404 301 161 655 559	488 38 20 221 97

DEATH BY CANCER IN GERMANY PER MILLION LIVING ON JANUARY 1

 		36		
 Year	Men	Women	Total	
1888 1900	373 574	445 648	818 . 1222	

Examination shows that the increase has been regular and progressive.

THE PROGRESSIVE INCREASE OF CANCER IN MUNICH 4
THE DEATHS IN MUNICH DURING THE LAST 39 YEARS

	TOTAL DEATHS		CANCER AND OTHER NEW GROWTHS		
Time Periods	In general	Per 1000 inhabitants	In general	Per 1000 inhabitants	
1871-1875	7229	40.4	136	0.8	
1876-1880	7563	35.4	207	1.0	
1881-1885	7485	30.4	265	1.1	
1886-1890	8363	28.3	330	1.1	
1891-1895	9846	25.8	428	1.1	
1896-1900	10724	23.9	609	1.4	
1901-1905	10845	21.0	756	1.5	
1906	9801	18.0	888	1.6	
1907	9965	18.1	905	1.7	
1908	10055	17.9	977	1.7	
1909	10060	17.6	890	1.6	

<sup>4</sup> A. Theilhaber and S. Greischer: Zeit. für Krebsforschung, ix, 530.

This shows the increase from 1871 to 1909. During the period 1871-1909, in Munich, the total mortality from all causes was greatly reduced. From 40.4 per thousand in 1871 it fell in 1909 to 17.6. During this period tuberculosis decreased from five per thousand to 2.3 per thousand.

The relative increase in cancer may thus be explained by the fact that the greatest reduction in the death rate was in tuberculosis and in diseases of childhood and early adult life such as diphtheria and typhoid fever.

According to Korber cancer statistics of Hamburg, 1908, per hundred thousand inhabitants from 1883 to 1903, death by cancer occurred as follows:

	In 1883	In 1903
Switzerland	102 58 50 53 44	131 99 93 85 74
	In 1893	In 1903
Germany	59	73

The author found 2213 cases representing the total deaths from cancer in the city of Munich from 1907 to 1909 inclusive, 784 cases of cancer of the stomach of which 410 were in women and 374 in men. The greater death rate from cancer of the stomach in women the author ascribes to the fact that more women than men reach the cancer age. More men die of diseases of childhood, and early in life from injuries, vocational diseases and tuberculosis. So far as cancer of the stomach is concerned he suggests that the trying of hot foods in pro-

cess of cooking may be a special factor in women. In the three years classified, 36 women cooks died from cancer, 16 of these had cancer of the stomach, 6 cancer of the uterus, 4 cancer of the mamma. With women other than cooks the figures are reversed. This author also shows that as age increases more women than men are affected with cancer. He finds cancer of the stomach more prevalent among the poor than among the well-to-do. In the year 1909, among the wealthy, cancer of the stomach represented only 30.3 per cent while in the poorer classes it represented 54.7 per cent.

Statistics of cancer in Denmark, by J. Fibiger and S. Trier,<sup>5</sup> taken in 1908 show the following: There were 13 cases occurring in individuals from 20 to 29 years of age, and of these 4 were cancer of the stomach. Of the 1135 living cases, 253 were cancer of the stomach (nearly a quarter of all cases, representing also the most frequent type of cancer). Of the 253 cases of gastric cancer 141 cases were in men and represented 31.9 per cent of all cancer in men. The remaining 112 cases in women represented 16.2 per cent of all cancer in women.

Carcinoma ventriculi is more common in the country than in the cities. In Copenhagen it represents one-fifth of all cases, and in the country, between one-third and two-fifths. This is extremely interesting when compared with the figures of cancer of the uterus. Only 51 of the total 155 cases of cancer of the uterus were found in the country, although the population in the country is about 200,000 more than that of the cities, the ratio being as 7 to 5.

Statistics for Finland covering all cancer cases from 1890 to 1907, show that in Finland cancer of the lip is much more frequent than in Sweden. Cancer of the lip

<sup>&</sup>lt;sup>5</sup> Zeit. f. Krebs., 275.

in Sweden is 4.2, in Finland 18.9. Cancer ventriculi, Sweden 45.3 per cent; Finland 24.2 per cent.

The general conclusions to be drawn from the foregoing statistics are that cancer of the stomach includes about 50 per cent of all cancer and that from 80 to 90 per cent of all cases of cancer occur somewhere in the digestive tract.

The ingestion of hot or irritating substances seems to predispose to cancer of the stomach.

Although more cases occur in men than in women, especially in certain districts, this difference is not marked if the figures from a wide extent are studied.

The predisposition to cancer at 70 is fifty times greater than at 30 years of age.

The Bavarian statistics show a striking difference in the frequency of the disease, comparing one district with another. In some countries the disease is most common in rural districts; in others, in the cities. In some localities more women than men are victims of carcinoma ventriculi; in others, more men than women are affected. The surprising frequency of cancer of the lip in some regions and of cancer of the esophagus in others is hard to explain.

I am indebted for the following description of the pathology of cancer of the stomach to my colleague, Dr. Herbert U. Williams.

## **PATHOLOGY**

"While other tumors of the stomach are occasionally encountered, carcinoma exceeds them in frequency so far as to be of supreme importance to the clinician.

"As is the case on many mucous surfaces, hypertrophy or hyperplasia of the mucous membrane occurs in the stomach as a result of chronic inflammation, and such hypertrophies sometimes assume a polypoid form and resemble tumors. (Fig. 56, page 576.) Growths of this nature are not, however, as common in the stomach as in some other locations under similar conditions, the uterus for example. The stomach may also be the seat of adenoma, and it is sometimes very difficult to decide whether a growth is an adenoma or a gastric polypus of inflammatory origin.<sup>6</sup>

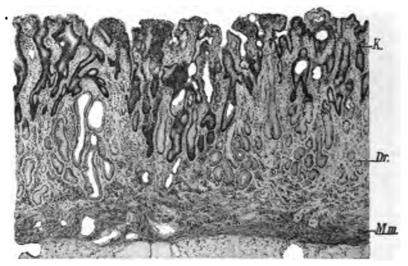


Fig. 29.—CANCER.

Surface growth of tumor cells, which send processes downward, growing between the normal glands of the mucosa.

(From Aschoff's Pathologische Anatomie, 2nd edition, p. 741; Fischer, Jena.)

"On the other hand it is equally difficult to draw a line sharply separating adenoma from adenocarcinoma. Precisely the same difficulty in classification occurs on many other mucous surfaces and in some of the latter cases the question is one of much greater moment than

<sup>&</sup>lt;sup>6</sup> This subject is well discussed by Cone in "Contributions to the Science of Medicine by the Pupils of W. H. Welch," p. 877: "Multiple Hyperplastic Gastric Nodules, etc."

in the stomach by reason of the greater frequency in such localities of glandular polypi and of adenoma. In general, infiltration of the wall of the stomach by carcinoma is the morphological characteristic that distinguishes it from adenoma. (Fig. 31.) It is natural to inquire whether or not carcinoma may sometimes find its start-

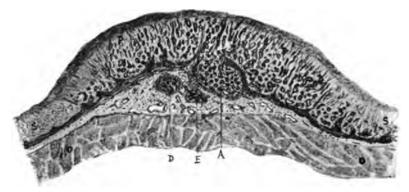


FIG. 30.—FLAT POLYP OF THE STOMACH WITH CARCINOMA ENGRAFTED. (Ribert, Das Karzinom des Menschen, p. 174—Bon, 1911.)

ing point in glandular polypi, but direct evidence is difficult to secure. However, the belief that such an origin is possible is quite general. (Fig. 30.) A similar question arises in connection with the relation between some cases of carcinoma and simple gastric ulcer, which is discussed below.

"The possibility that fragments from benign gastric polypi may appear in stomach washings was suggested by a specimen from a middle-aged man submitted to the writer. The shreds of tissue in this case showed glands of a distinctly abnormal form and closely resembling an illustration of a fragment of carcinomatous tissue in stomach washings given in a well-known work. The patient in my case is now in excellent health although about seven years have elapsed.

"Cancer of the stomach is quite rare before the thirtieth year, and not common before the fortieth.

"From the figures of Welch and of Orth and the statements of other observers it appears that about 60 per cent of the growths are in the pyloric region, 10 to 20 per cent on the lesser curvature, about 10 per cent in the cardia, and the remainder in other parts of the stomach.

#### ANATOMY

"The appearance of carcinoma of the stomach varies considerably in different cases. Frequently the tumor has the form of a large papillary or fungus-like mass protruding into the cavity of the stomach, although the microscope will show that the wall of the stomach is infiltrated. Other cases are characterized by a ring-like growth, others simply by a diffuse infiltration of the wall of the stomach. Hypertrophy of the muscular

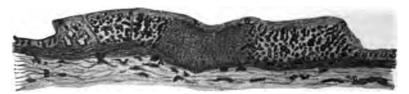


FIG. 31.—BEGINNING CARCINOMA OF THE STOMACH. (Ribert, Das Karzinom des Menschen. Bon. 1911.)

layers may take place. A new growth of connective tissue is a common occurrence and may give rise to the impression that one is dealing only with fibrous induration if the growth on the surface is not conspicuous. (Fig. 32.) The infiltration of the carcinoma proceeds through the wall of the stomach by way of the lymph spaces and lymphatic channels. In the end it reaches the peritoneal surface. The peritoneum may now become extensively involved. Other organs may also be invaded by direct

extension, largely after the formation of fibrous adhesions, as the transverse colon, the liver, the spleen, the pancreas and the diaphragm. Carcinoma at the cardia may extend along the inner surface into the esophagus; more rarely, carcinoma at the pylorus extends into the duodenum. Ulceration occurs frequently, being the re-

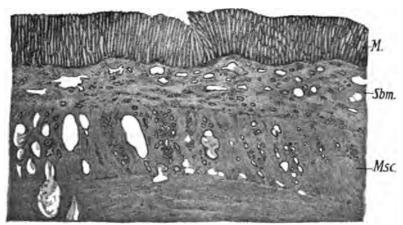


FIG. 32.—ADENO-CARCINOMA OF THE STOMACH.

Continued growth of the cancer process in the lymph vessels of the submucosa. (In such a case the growth could not be recognized early even if the stomach were opened and inspected.)

(From Aschoff's Pathologische Anatomie, 2nd edition, p. 740; Fischer,

(From Aschon's Pathologische Anatomie, 2nd edition, p. 740; Fischer Jena.)

sult of degeneration and necrosis in the new growth, to which the action of the gastric juice may probably be added in some cases. Perforation into the peritoneal cavity, into neighboring organs and even through the anterior abdominal wall may occur.

"Hemorrhage, especially from the softer, protruding growths, is not uncommon. The erosion of small vessels producing the so-called "coffee grounds" contents is frequent; hemorrhage from large vessels is rather exceptional.

"Pyloric stenosis, with all its consequences, often occurs as a result of carcinoma. "Chronic gastric catarrh of the remainder of the stomach is also very common, and may be responsible for the

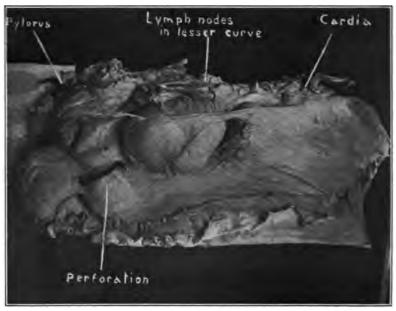


FIG. 33.—CARCINOMA OF THE PYLORUS.

Both perforation and lymphatic involvement are shown. The outside of the stomach is seen, the black tapes being in the esophageal and pyloric ends of the stomach, and the perforation just below the large lymph node. This was the cause of fatal peritonitis.

lack of free hydrochloric acid that is a symptom of so many cases of carcinoma of the stomach.

#### HISTOLOGY AND CLASSIFICATION

"The cells that constitute the essential part of the growth arise in all cases from the epithelial cells of the stomach, probably chiefly from the cells of the glands. The varieties into which carcinoma of the stomach may be divided are based upon the microscopic structure as well as the different gross appearances presented by different cases. They are connected with one another by forms of an intermediate character. The varieties may be classi-

fied as adenocarcinoma, medullary, scirrhous, and gelatinous. Squamous-celled carcinoma beginning in the lower end of the esophagus and extending into the stomach, is sometimes added to these.

"Adenocarcinoma (cylindrical-celled carcinoma) usually projects into the cavity of the stomach, commonly near the pylorus, as a soft and often papillomatous growth. Microscopically it consists, in part at least, of tubular structures lined with cylindrical epithelial cells, less regular in size and form than normal glands, and infiltrating the deeper layers of the wall of the stomach. Usually in some part of the growth the lumina of the tubes are obliterated and are completely filled with cells, and the picture of ordinary carcinoma prevails.

"The terms medullary and scirrhous are sufficiently well known to make explanation of these varieties superfluous. Naturally it is the scirrhous variety that is especially prone to produce narrowing at the pylorus or to convert the pyloric end of the stomach into a firm and unyielding tube; it is also inclined to spread over a considerable portion of the wall (so-called cirrhosis). The softer medullary variety is more likely to produce masses that project into the stomach cavity, and often ulcerates.

"Gelatinous carcinoma (the so-called colloid cancer) may involve a considerable extent of the stomach wall. It leads to a thickening that has a characteristic translucent appearance. Microscopically, the collections of epithelial cells are found to be replaced in whole or in part by structureless or stringy gelatinous material containing remnants of cells. This material appears to be mucin or some related substance produced by the epithelial cells as a secretion or a product of their degeneration; possibly some of the stroma may undergo a similar change.

A glandular arrangement of the epithelial cells is frequently recognizable in this form of carcinoma.

## **METASTASES**

"Secondary involvement of the lymphatic glands usually takes place early, notably in the glands of the lesser curvature; the portal, retroperitoneal, and even more distant glands, as the supraclavicular, may be involved.

"Although metastases in other organs may also occur, by all means the most important location for them is the liver, which is said to contain secondary tumors in 25 to 30 per cent of the cases if not more. It is possible for a metastatic growth to be larger than the primary tumor. Thus in the liver, an erroneous diagnosis of primary carcinoma may be made, during life, when one is in reality dealing with a tumor secondary to a carcinoma of the stomach. The transfer of the growth from the stomach to the liver usually takes place by way of the portal vein. The enlargement of the liver may be enormous; in one of the writer's cases it weighed twelve pounds."

## CLINICAL HISTORY AND SYMPTOMATOLOGY

The clinical manifestations of cancer of the stomach vary, to some extent, with the character and location of the growth.

Classification as to Character.—As to character, the cancer may be: (1) vegetative, the growth projecting into the stomach; (2) infiltrating, extending into the deeper layers instead of involving merely the superficial ones; (3) ulcerative; (4) hard and contracting; or (5) it may be considered to be chronic, latent or masked.

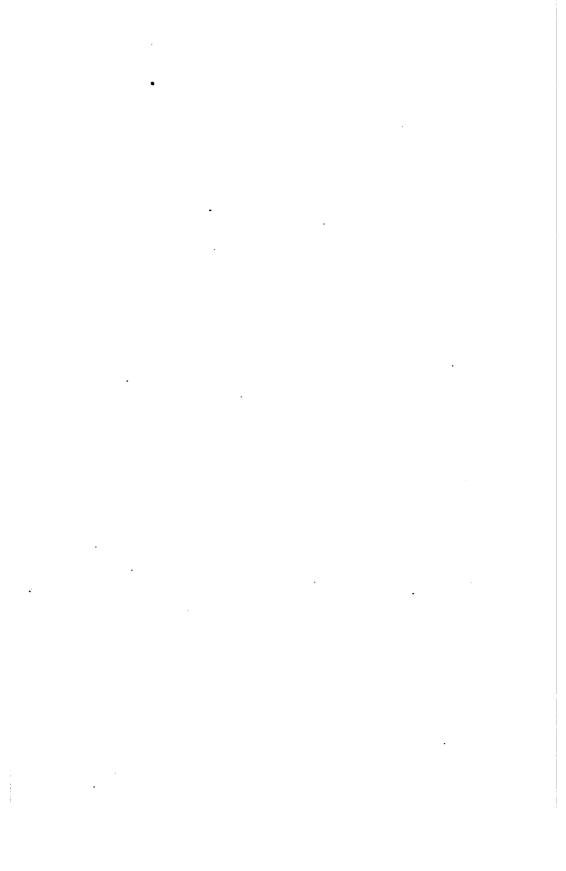
Classification as to Location.—(1) The disease is in a few cases diffuse, invading the entire lining of the stom-



Plate No. III

CARCINOMA INVOLVING ESOPHAGUS AND CARDIAC END OF THE STOMACH.

Section by B. T. Simpson. The specimen is interesting and rare in showing the growth of flat epithelium of the esophagus, columnar epithelium of the stomach and areas which show a mixed type. a, Proliferation of flat epithelium; b, Columnar epithelium; c, Mixed flatand columnar epithelium.



ach, apparently in equal degree, and infiltrating toward the outer coat. It forms in this type a large epigastric tumor. As a result of the rigidity caused by the infiltration of the walls, the stomach becomes a cancerous shell. The cardiac and pyloric orifices, although stiffened, may remain open.

- (2) The disease begins along the anterior or posterior wall of the stomach, or at the lesser curvature.
- (3) A precocious type in which the growth invades the cardia.
  - (4) Cancer of the pylorus or the pyloric region.

CANCER INVADING THE INTERIOR OF THE STOMACH.-Even when extensive, the so-called cancer en nappe, may cause comparatively little suffering, provided that the cardia and pylorus remain open and that the stomach does not greatly contract. Pain may be absent until the peritoneum is attacked or until unnatural tension is produced through adhesions to surrounding parts. ing may be absent; in fact in some cases the rigidity of the stomach wall renders vomiting extremely difficult. As the carcinomatous process advances, the stomach becomes more and more contracted, hence the containing power becomes lessened, and there sometimes develop symptoms that are misleading, that is, those closely resembling cardiac obstruction with esophageal symptoms. This type has been described by Cade 7 of Lyons. There is upon taking food a sensation of fullness with regurgitation immediately after eating, at first of solid foods only but later in the disease even of liquid food. Although the clinical picture is that of cardiac stenosis, yet upon the passage of the stomach tube, the cardia is found to be patulous. Attempts to inflate the stomach, either with air or fluid, reveal the actual condition. Radiography con-

<sup>&</sup>lt;sup>7</sup> Arch. des Mal. de l'Appar. Digest., 1911.

firms the conclusion that the stomach is contracted and therefore incapable of receiving more than a modicum of food. The fact of frequent regurgitation is not of itself sufficient ground for the diagnosis of cancer of this type. Regurgitation may result from infiltration and hence irritation in the neighborhood of the cardia, although the passage is unobstructed. Also, it may result from cancer of the lesser curve or even of the pylorus. I have not met with a case causing the esophageal symptoms described by Cade and confirmed by others.

In addition to regurgitation or vomiting, gastric distress and a sense of weight in the epigastrium are the most important local symptoms of diffuse cancer of the interior of the stomach. The general symptoms include complete anorexia, early distaste for meat, rapid loss of weight and cachexia.

CANCER AT THE ANTERIOR OR POSTERIOR WALL OF THE STOMACH OR ITS LESSER CURVATURE.—When cancer occurs in these regions, provided the disease does not give rise to a projecting mass but limits itself to infiltration of the gastric walls, the local symptoms may be inconspicuous until the growth is sufficiently extensive to produce a definite induration that can be felt upon palpation. When the symptoms appear they are those of local irritation or of indigestion. There are significant decline in gastric secretion, localized distress after eating, eructations of gas and disagreeable regurgitations. Nausea and sometimes incessant vomiting may be early symptoms. When the cancer is limited in extent the cachexia may be slight. The degree of emaciation depends upon the ability to take food. When the growth is vegetative and projects into the stomach, local symptoms become conspicuous There is soreness, distress and pain, especially when vomiting occurs. Even when the growth is small,

there is likely to be hemorrhage, often slight, sometimes abundant, with hematemesis and moderate melena.

CANCER AT THE CARDIAC END OF THE STOMACH.—Cancer at the cardiac end of the stomach makes itself known by inability of the patient to take foods on account of accompanying spasm of the cardia or esophagus. These spasmodic symptoms occur early in the disease before the growth is large or obstructive and but for the inaccessibility of the cardia because of its relations to important surrounding structures, it would be possible to rescue many cases by surgery. The symptoms at first may be confined to dysphagia, the spasm occurring at the pharynx or at the cricoid. The symptomatology of this type of cancer and its differential diagnosis are more fully considered in the special chapter devoted to the diseases of . the esophagus. It need only be said here that eating becomes difficult and painful, spasm and regurgitation assume great importance, gastritis supervenes and, although the cancer may not extend rapidly, the patient rapidly grows worse.

Cancer of the Pylorus or the Pyloric Region.—Cancer located in the pyloric region occurs in from 60 to 75 per cent of cases of gastric cancer and with the exception of the cardia is more promptly attended with symptoms than when it appears in other regions of the stomach. Sometimes indeed spasm of the pylorus occurs as an extremely early manifestation, and this is particularly true when gastritis is prominent. In case the disease has not been preceded by gastric ulcer and in case its symptoms do not simulate some other constitutional disease, in other words, when it attacks a person previously well, an early diagnosis may be possible and, fortunately, this is the region of the stomach upon which a surgeon may operate with the greatest probability of success.

The symptoms of cancer at the pylorus are those of pyloric irritation with spasm. There is the beginning of motor insufficiency as shown by eructations which remind the patient of a previous meal; nausea is likely to follow, and vomiting is often encouraged by the patient since it brings at least temporary relief. In a group of cases there is intractable vomiting and in others there develop symptoms that suggest cardiac obstruction. When the growth has been preceded by ulcer, there is almost invariably a rapid and significant disappearance of the preceding highly acid gastric secretion; lactic acid appears in the stomach contents, and usually there are found the long branching bacilli described by Oppler and Boas. beginning these symptoms are sufficiently striking to attract attention and to create the suspicion of cancer. It is in these cases that careful laboratory study of the dis-. ease, the examination of the stomach contents, stools and blood are essential.

Cancer of the pylorus is frequent. By irritation it causes pyloric spasm early in the disease. We know also that cancer most often occurs at middle age or later. Knowing the age at which to expect the disease and the early symptoms of the pyloric type, it would seem that we might recognize pyloric cancer early in its development. However the pylorus is unfortunately the region most often affected by gastritis and peptic ulcer. the region most subject to sympathetic disturbances which reflexly announce diseases extrinsic to the stomach, such as diseases of the gall-bladder or the appendix. Surgeons complain because cases of carcinoma are not referred to them early enough to make good results possible. after year we have this censure repeated. The statement indisputably is well grounded, yet the fault lies not so much with the diagnostician as with the inherent difficulty

of the problem. As Boas remarks, the internist cannot be held responsible for cases before they come for examination and it is a fact that usually the patient does not seek medical advice until the disease, from a surgical point of view, is far advanced. Yet there is no excuse for the delay and indecision displayed in the management of cases in which a comparatively early diagnosis is possible.

So far as relates to pyloric cancer the following may be said to represent an early picture of the average case. There is disturbance or falling off of appetite, a lack of zest for certain foods, especially for meats. Motor symptoms develop gradually, sometimes suddenly. The patient recognizes them by the "belching of gas" and by the "repeating" of food. These symptoms, temporarily relieved by diet and the use of simple household remedies, recur increasingly without satisfactory explanation. Very soon the patient experiences more pronounced gastric distress and more persistent uneasiness, usually the expression of advancing stasis. This stasis is often occasioned by gastric over-tonicity and moderate spasm of the pylorus.

It is at about this time that the patient thinks it wise to consult a physician, although if befogged by "mental science" he may delay still longer. By now the disease is already considerably advanced as is shown by the findings. There is a slight cachexia and some falling off in the red blood cells. Generally there are marked changes in the gastric chemistry. The total acidity of the stomach contents is diminished and the acidity depends less upon free hydrochloric acid than is usual. Organic acids, especially lactic acid, are present in excess. The bacteria are not those of the normal stomach.

The differential diagnosis between the various forms

of pyloric stenosis is of great importance because of the question of possible carcinoma. We have to consider the following contingent conditions:

(1) Duodenal ulcer; (2) Pyloric ulcer; (3) Stenosing gastritis; (4) Primary cancer of the pylorus; (5) Cancer grafted upon pyloric ulcer; (6) Functional hyperchlorhydria followed by ulcer or cancer. With any of these we must expect to find motor insufficiency of the stomach. Having then symptoms of motor insufficiency we must answer the question, upon which of the numerous causes do they depend? With pyloric or duodenal ulcer there is usually present pain developing several hours after eating, which is temporarily relieved by taking food. This so-called "hunger pain" is not limited to duodenal ulcer, as has been pointed out in preceding pages. belongs equally to ulcer located anywhere in the pyloric area, and may be present in stenosing gastritis, or hyperchlorhydria, but it is not usually present with cancer, even when the cancer is one growing upon ulcer, although to this there are exceptions. With ulcer there is usually hyperchlorydria; in cancer, hypochlorhydria and lactic acid. When cancer grows upon ulcer, as a rule there is a significant change from a high to a low secretion of hydrochloric acid. Gastritis sometimes depends upon syphilis, in which case the secretion occasionally remains high, while in other respects the case resembles cancer. The differentiation may be made by the discovery of other manifestations of syphilis and the finding of the characteristic Wassermann reaction. Even in the absence of this specific test one may be governed by the other signs of the disease and by the prompt relief which follows the hypodermic injection of mercury, the intravenous injection of salvarsan, and the taking of the iodid salts by the The presence of tumor does not necessarily mean mouth.

cancer, for the tumor may depend upon inflammatory induration, perigastritis, or syphilitic granuloma.

Prof. Le Dentu<sup>8</sup> of Paris has drawn attention to the importance of distinguishing between inflammatory tumors of the abdomen and malignant growths.

Case No. XXI Inflammatory Tumor Resembling Cancer.—A middle-aged woman, a family cook, entered my ward with lobar pneumonia and pleurisy. Her recovery was delayed because of cystitis occasioned by an overdistended bladder and catheterization. Later there was discovered a deep-seated tumor, but slightly movable, irregularly rounded in form, as large as the fist, located just below the umbilicus. It was moderately tender upon pressure and slowly increased in size. There was persistent temperature, oligocythemia and a leucocytosis of 16,000 which might have depended on the infected urinary There was considerable mucus in the stools. patient complained of lumbar backache. Dr. King, on pelvic examination recognized a small uterine fibroid, but above this was a mass, the nature of which he was uncertain, but which it was thought might be inflammatory. Soon afterwards this diagnosis was confirmed by a discharge of pus per rectum, after which the backache lessened and the fever decreased. For several months there was more or less purulent discharge, during which time the tumor subsided. At the end of a year it had quite disappeared and the patient was well. This tumor was for a time supposed to be cancer, possibly gastric. Its etiology was uncertain. Probably like the case of Le Dentu it was an instance of pericolitis with involvement of the mesentery.

Deep-seated inflammatory tumors may transmit the aortic pulsations and, being associated with excruciating

<sup>8</sup> Bull. Méd., Nov. 17, 1909.

lumbar pain, may well be mistaken for abdominal aneurysm. Likewise certain cases of cancer of the stomach may cause confusion by transmission of aortic pulsation.

In distinguishing cancer from other diseases the antitryptic reaction alone has but little diagnostic value; however, when it is associated with a marked hemolytic action of the serum, its value is augmented. Some observers believe that when there is found simultaneously present a notable increase of the antitryptic reaction and of hemolytic action of blood serum, it may be assumed that these reactions depend upon cancer. Of this I am not convinced although experience warrants further trial.

Radiography may materially assist in reaching a differentiation. The shadow corresponding with a neoplasm is of diminished density, it is vague and its borders are irregular. Persistence of a small and sharply localized shadow when the stomach has for the most part discharged the bismuth is strongly suggestive of ulcer in contradistinction to cancer. (See Radiograms XIX and XX.)

Cancer Growing upon Ulcer—Ulcus Carcinomatosum.

—It is quite generally believed that some cases of carcinoma of the stomach originate at the edges or in the scars of simple peptic ulcers. Such a belief is not unnatural, considering the numerous analogies presented by other parts of the body where carcinoma appears after old inflammatory processes and in scars, such as carcinoma of the skin developing in the scars left by burns.

Hauser 9 in his well-known work contended as long ago as 1883 that growth and proliferation of the gastric glands may be shown in the scars of gastric ulcers, and

<sup>&</sup>lt;sup>9</sup> Das chronische Magengeschwür, sein Vernährbungsprocess, und dessen Beziehungen zur Entwicklung des Magencarcinoms. Leipzig, 1883. Accessible to the writer only in his brief communication in the *Pruger Med. Woch.*, viii, 136.

that these glands may grow into the submucosa. It was thought possible that such growths might increase and become carcinomatous.

Undoubted carcinomata are from time to time found growing at the edge of a partially healed simple gastric ulcer. Orth points out that inasmuch as carcinoma itself frequently ulcerates, an ulcer may be the result and not the cause of the tumor, and that in some cases it may be difficult or impossible to decide which was the primary

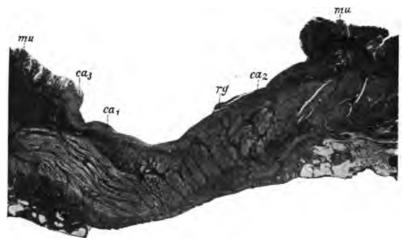


FIG. 34.—CANCER GROWING ON ULCER.

Invasion by cancer on margin of old ulcer.

(Verhandlung der Deutschen Path. Gesell., 1909. Article by Herr Verse of Leipzig, p. 376.)

process. When the ulcer is surrounded by carcinomatous masses, the carcinoma probably preceded the ulceration. When a typical, simple gastric ulcer shows a carcinomatous tumor only on one side, the tumor is probably secondary to the ulcer.

It is universally admitted that cancer may develop from the scar of preceding ulcer, as pointed out by Orth, Aschoff and others; a carcinoma may ulcerate and at that point assume the appearance of chronic peptic ulcer. It is at present a warmly debated question as to how frequently carcinoma is introduced by ulcer.

The experience of the Mayo clinic as reported by Wilson and MacCarthy indicates that gastric cancer springs from chronic ulcer in the majority of cases, and the experience of Moynihan is corroborative of this view. Most surgeons, while not prepared to accept the frequency of this relationship of cancer and ulcer, nevertheless agree that it exists more often than the internist will admit.

Those internists who see many cases of ulcer and whose opinion, based on final results, must be considered, believe that not more than five to fifteen per cent of cases of cancer of the stomach begin as chronic peptic ulcer. The probable reasons for this discrepancy are as follows:

- (1) The character of cases seeking relief at the Mayo clinic is peculiar. Perhaps this is owing to special tendency to cancer in the region which supplies this clinic; perhaps it arises from the fact that a larger proportion of unusually troublesome cases are referred there.
- (2) The basing of diagnosis of cancer on histologic examination is not without possible misrepresentation. Many have discarded the practice of making immediate microscopic examination of frozen sections, regarding them as unreliable.

Pathologists of great experience decline to commit themselves as to malignancy of many indurative formations involving the pylorus although in structure they closely resemble cancer.

We must regard the general features of the case including the history as important, if not necessary, in deciding the nature of certain specimens.

In very rare instances all means of diagnosis prove to be erroneous; an occasional case is reported in which the course of the disease has indicated cancer, in which the tumor has been removed, examined and found to have the structure of carcinoma, in which the growth has recurred, then receded and the patient ultimately has regained health.

The course of chronic ulcer is usually marked by occasional recession or even disappearance of symptoms, whereas the symptoms of ulcus carcinomatosum usually continue and grow worse.

In chronic peptic ulcer the pain often subsides after eating, but when carcinoma is grafted on ulcer the taking of food increases the suffering.

Cancer growing upon peptic ulcer has at times an insidious beginning. Slowly and at first almost insensibly, there takes place a modification of gastric function. times the secretion of hydrochloric acid may remain at a high point even when the growth has become palpable upon physical examination. If we find a tumor coincident with even slight decrease in the secretion of hydrochloric acid and accompanied by continuing signs of pyloric irritation, it is wise to assume that its nature is malignant. In other cases cancer betrays its invasion of an ulcer by abrupt changes in the symptoms. Of most importance among these is great increase of irritability without a corresponding increase in the hyperacidity. Unfortunately, it sometimes happens that the total acidity in the stomach contents rises, a fact that depends upon obstruction at the pylorus and consequent delay in discharge of the stomach contents, or the development of the Reichmann syndrome. These cases are misleading. creased irritability of the stomach might wrongly be attributed to the rising hyperacidity and this hyperacidity might be mistaken for a renewal of localized gastritis, with swelling and spasm from a re-awakening of the ulcer process. Errors of this kind are bound to occur and offer a cogent reason for the surgical removal of these masses of inflammatory tissue before they have undergone malignant degeneration. The history of the ensuing case sets forth the difficulty that is sometimes encountered.

Case No. XXII Long Continued Chronic Dyspepsia, Recurring Peptic Ulcer, Finally Beginning Cancer.—Mr. H., aged 50, had suffered from chronic ulcer with characteristic history extending over a period of two years. He was under my observation for about a month.

There was high gastric acidity, and lactic acid and Oppler-Boas bacilli were absent. There were pain, occasional presence of occult blood in the stomach contents and its persistent presence in the stools. The absence of palpable tumor, except slight resistance over the pyloric region, led to the belief that we were not dealing with cancer but with pyloric or duodenal ulcer and perigastritis. Meantime the stomach was emptied at night, bismuth introduced and the stomach left at rest until the morning. Before this practice the fasting morning stomach commonly contained nearly a liter of stagnant contents. Great relief followed this treatment. With appropriate diet, antacids and sedatives there was progressive improvement; the patient ceased to lose weight and felt that he was recovering. After two weeks there appeared signs of returning stagnation. I then proposed operation, which was done a week later. There was found a large cartilaginous ring at the pylorus; there was considerable perigastritis with adhesions to the omentum. Small areas of ingrowing tissues were present at and below the pylorus, one of them being more than an inch below the pylorus on the posterior fixed wall of the duodenum. Palpation discovered several large retroperitoneal lymph nodes. The case was carcinoma engrafted upon ulcer, as was shown by histological examination.

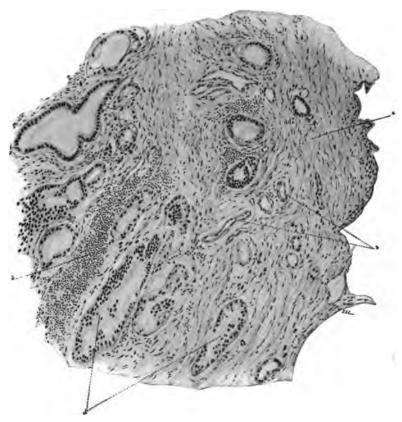
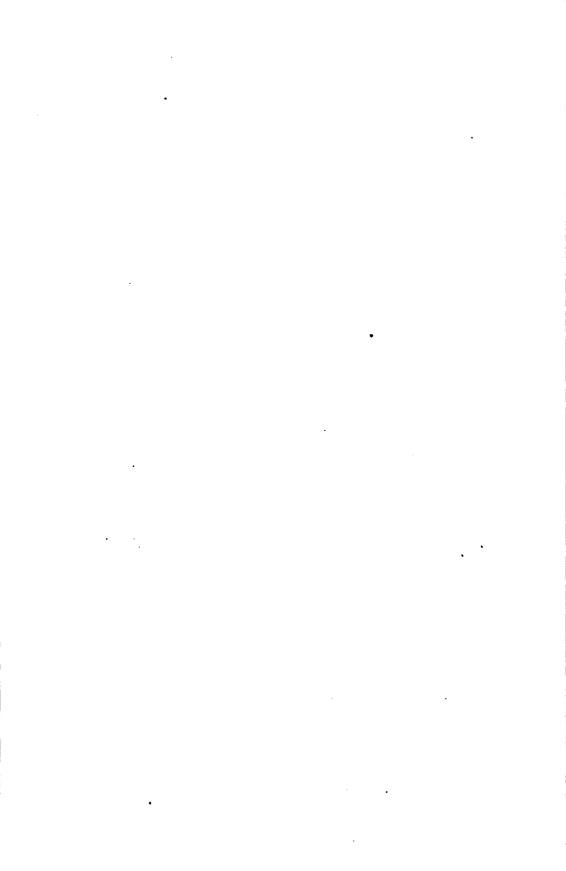


Plate No. IV

CANCER GROWING IN SCAR OF PRECEDING ULCER.—Tissue removed from case No. 22. Section by B. F. Schreiner. a, Lymphatic tissue; b, Blood vessels; c, Scar tissue; d, Carcinomatous infiltration.



The duodenum was too much involved to admit of resection of the diseased tissue; a gastrojejunostomy alone was practicable, and this operation was performed successfully.

It seems probable that carcinoma had begun in this case before the patient came under my observation. standing, with careful medical treatment he not only gained in weight but in most other ways showed improvement. He ate with less reserve than at any time during the preceding year and the gastric irritability came to be only moderate. Previous treatment had probably been irregular and not well ordered. The deceptive improvement doubtless was the result of the establishment of treatment that met the indications of the case. Had the patient from the start been under the same regime instead of temporary improvement with the onset of the cancer, there might have appeared more definite indications of the dis-This history shows that the early diagnosis of cancer, when it begins in the scar of an old ulcer, is difficult and at times impossible.

Histological examination of the tumor removed in the above case showed that the mass was for the most part made up of scar tissue, the result of the ulcer. With the exception of a very small area it was free from cancerous infiltration.

It must be remembered, however, that hyperchlorhydria of functional origin is a reality, and is often accompanied by pyloric spasm. The cause of this aberration is not always apparent. It may depend upon some derangement of internal secretions exciting abnormally the gastric innervation. Some patients have recurrence of this condition during a lifetime, very probably without any local disease at the pylorus. These cases are not exempt either from ulcer or cancer, and when either of these develops

the symptoms may be confusing. Patients who suffer from functional hyperchlorhydria with spasm or from other functional gastric disturbances should be instructed to report immediately the appearance of any unusual symptoms. If ulcer develops, the spasmodic element is intensified and traces of blood may be discovered in the stomach contents. If cancer develops, the gastric irritability becomes constant, usually without an increase in the hydrochloric acid, there may be traces of blood and the symptoms of motor insufficiency are more impressive than the actual stagnation would account for.

## UNUSUAL FORMS OF CANCER OF THE STOMACH

The types of the cancer, as already described, depend for their manifestations partly upon the location, character and extent of the growth. Besides these usual forms there are other expressions of cancer of the stomach less typical; latent cancer and certain rare forms that remain to be considered briefly. The unusual forms have been described and classified recently by Rousseau.<sup>10</sup>

However, any classification must necessarily remain incomplete, for we are constantly meeting with new and unexpected forms.

Important are those cases in which occur striking derangement of the general health, but with slight local evidence of the disease and an absence of positive laboratory proof. This is illustrated in the following case:

Case No. XXIII. Mental Depression, Anorexia, Vague Abdominal Distress; A Remarkable Absence of Local Symptoms.—D. L., aged 56, a farmer previously well, had experienced irritability of the stomach for five years and for two months marked anorexia. He was despondent, at times giddy, and complained of continual vague pain at

<sup>10</sup> Arch. des Mal. de l'Appar. Digest., Aug.-Sept., 1912.

the ensiform, radiating to the back. He had lost twentyseven pounds in weight.

He was given an Ewald test breakfast. One hour afterwards there was aspirated 70 c.c. of normal contents; total acidity, 40; free hydrochloric acid, 26 (.09 per cent); combined chlorids, 8; acid salts, 6; no lactic acid; no occult blood; no mucus; no Oppler-Boas bacilli. There was good motility.

The stools were normal including absence of occult blood.

The blood showed: hemoglobin, 88 per cent; erythrocytes, 4,608,000; leucocytes, 7,600; polymorphonuclears, 72.0; small lymphocytes, 20.4; large lymphocytes, 3.06; eosinophiles, 0.0; basophiles, 0.0; transitionals, 4.0.

Red cells satisfactory in shape and size. Wassermann reaction, negative.

The antitryptic reaction was  $3\frac{7}{40}$ , i. e., within the normal range.

The complement duration reaction showed: A. with non-specific antigen, not positive; B. with cancer antigen, negative; C. with gonorrhoea antigen, negative.

Hemolytic test:

- A. No abnormal hemolysis was exerted by the serum on the corpuscles of other individuals.
- B. The corpuscles were not abnormally resistant, being hemolyzed by sera of known cancer cases.

None of the tests indicated even a suspicion of cancer. The diagnosis was withheld and the patient treated as a neurasthenic, with forced alimentation, rest and suggestion. There was gain in weight, decrease of pain and improvement in *morale*. The patient having returned to his home, his condition became worse. He then submitted to an exploratory incision. There were found in the stomach scattered plaques shown by histological exami-

nation to be adenocarcinoma. Two of these plaques were about the size of a half-dollar and were located on the anterior surface of the middle zone of the stomach. They did not involve the mucosa. There were similar plaques on the surface of the liver, on the omentum, intestine and appendix.

I am unable to see how a diagnosis could have been made in this case. The picture was that of general debility and neurasthenia; there was constant complaint, querulousness, depression, a state suggesting mental disease.

Cases may present fever only as a prominent symptom. Persistent high temperature attended with regular remission or intermission is encountered, sometimes without signs of local disease. Fever is, of course, to be expected in cases complicated with pyogenic infection, but in the type referred to no satisfactory explanation of the hyperthermia is found at autopsy.

Progressive anemia may occupy the clinical field largely to the exclusion of other evidences. A severe secondary anemia is to be expected, but the clinician is easily led astray when the blood picture is that characteristic of pernicious anemia or that produced by intestinal parasites. The following case illustrates some of these features:

Case No. XXIV. Cancer of the Stomach With Profound Secondary Anemia; Good Appetite; Intermittent High Temperature With Chills; Leucocytosis; Large Number of Amebae Coli in the Stools; An Unusually Movable Tumor in the Midepigastric Region.—Mr. S., aged 50, river boatman, began suffering from stomach symptoms twelve months ago. He complained of moderate epigastric distress and profound weakness. The stomach contents showed after an Ewald test breakfast

110 c.c., total acidity, 14; combined chlorids, 8; HCl, absent; lactic acid present; peptone and pepsin present; a little mucus and a marked occult blood reaction present.

The blood examination showed: hemoglobin, 28 per cent; red cells, 2,320,000; white cells, 10,400 (also 16,200); polymorphonuclears, 86 per cent; small lymphocytes, 9.2 per cent; large lymphocytes, 2.4 per cent; eosinophiles, 0.4; basophiles, 0.4 per cent; transitionals, 1.2 per cent.

The stools which appeared well digested, contained an enormous number of amebae coli, yet there was no evidence that their presence modified the symptoms. The temperature showed an irregular, intermittent elevation, rising from a subnormal point to 105° F. There was no vomiting, but considerable emaciation. At times the stomach contents was like that of achylia gastrica and . would have caused confusion excepting that a number of critical examinations were made. Occult blood was discovered, but that not uniformly. There was slight evidence of the presence of gastric proteolysis. The disparity between the red count and the hemoglobin, the character of the stomach contents, the fever, leucocytosis, anemia and tumor might all have been explained by cancer of the stomach.

There was marked antitryptic reaction, but no hemolysis.

In this case the complaint was of debility, such as might be explained by the anemia and fever; there was singular absence of gastric symptoms. With the leucocytosis, the high temperature and the presence of amebae coli, it would have been possible, excepting for the tumor, to make diagnosis other than cancer. A surgical exploration revealed a tumor the size of an egg on the posterior wall near the lesser curvature in the pyloric third of the stomach. The pylorus was not involved. The stomach

was freely movable. There was no ulceration or mixed infection to explain the fever. A movable tumor is less exceptional than may be supposed. Several times I have met with instances in which a pyloric growth occupied a position sometimes in the left upper quadrant, sometimes in the right, sometimes below the umbilicus. Osler describes a case in which, owing to gastroptosis, the tumor was in the pelvis.

In one case I found great diminution of the red cells with a high hemoglobin index. There were present nucleated red cells and some red cells large in size, others small and deformed. Rousseau describes certain rapidly fatal cases having metastases into the bone marrow in which occurred still more misleading blood changes, the presence of numerous myelocytes and an increase in leucocytes. In such cases the spleen and liver become large, there is characteristic bone tenderness and subcutaneous hemorrhages. The condition is commonly taken to be splenomyelogenic leukemia.

Sometimes accompanying the anemia, even without unusual blood failure, there develops a general anasarca that is misleading. Osler and McCrae found this in 10 out of 150 cases, not including local edema resulting from thrombus; Rousseau notes general edema in 4 out of 526 cases.

Metastases to the kidney serve to explain some of these cases of dropsy; involvement of the thoracic duct explains others. There remain a few rare cases not easily explained. Edema of one lower extremity, usually the left, may depend upon thrombus caused by unsuspected cancer of the stomach.

Ascites alone is far more frequently met with than general dropsy. It depends upon portal obstruction, metastases to the liver, perigastritis, chronic peritonitis and oc-

casionally gross lesion is found to account for the ascites.

Osler and McCrae report 8 cases of ascites out of 150 cases of gastric cancer, 6 of which showed secondary metastases in the peritoneum with none in the liver, and two metastases in the liver and none in the peritoneum. A metastasis to the ovary is likely to give rise to ascites, even when the growth is small. Unless the gastric symptoms are prominent the true cause of the ascites usually evades discovery until the fluid is withdrawn, when a tumor may be palpable.

Too much reliance should not be placed upon the presence of cachexia, hematemesis and melena in diagnosing gastric carcinoma, for all three may result from chronic uremia. Usually, in uremia, the hemorrhage depends upon toxi-infectious ulceration which, although occurring often in the colon, may take place in various parts of the digestive tract. Hemorrhage not limited to the digestive tract showing at autopsy no lesion to explain it, may be attributed to lowered coagulability of the blood and to vasodilatation, the result of toxemia. Not only are cases of uremia mistaken for cancer of the stomach, but supposed uremia may turn out to be cancer of the stomach.

Primary cancer of the liver is so exceptional that we usually expect to find that the stomach is the original seat of the disease. Secondary cancer of the liver behaves so differently in different cases that a wrong diagnosis is often made. With inconspicuous stomach symptoms there may be intense jaundice, intermittent jaundice, or only a slight icteric appearance, or there may be ascites either alone or associated with jaundice; or there may be a massive liver without either ascites or jaundice; or a deformed, bosselated or indurated liver, closely resem-

bling that produced by syphilis or by cirrhosis. With a diseased, enlarged or deformed liver, we must not only closely consider the diagnosis of cancer but also primary gastric cancer.

The jaundice of cancer according to Rousseau, is produced most often (15 out of 25 cases) by compression of the biliary passages outside the liver, that is, of the hepatic or common duct. Enlarged lymph nodes, adhesions or infiltrations, and enlargement of the pancreas may lead to the compression. In 7 out of 25 cases the jaundice arose from metastasis in the liver. When thus produced the cholemia is less intense, and the stools usually show the presence of bile. In 4 cases the icterus was caused by invasion of the biliary ducts. In such cases the jaundice may or may not be marked, depending on the ex-The frequency of hepatic metastasis tent of obstruction. is debatable. Osler and McCrae found it at autopsy in about 23 out of 150 cases. However, most authors find a larger proportion; and it should be remembered that the liver may be invaded even when it does not show macroscopic lesions, for lesions too small to be made out with the eye are often found microscopically. Moreover, before characteristic cellular changes have occurred there may be clinical evidence of hepatic insufficiency.

The presence of gastric cancer is at times obscured by persistent intestinal symptoms, which are usually caused by metastasis in the intestine or by adhesions. With no discoverable lesions of the intestine there may yet occur functional disturbances that are misleading. The most frequent intestinal symptom is diarrhea, either lienteric, watery, mucoid or blood-stained. This is attended with marked discomfort and often distress. It is aggravated by taking food, is accompanied by toxemia and is but temporarily relieved by purging or fasting.

Another group of intestinal symptoms is due to partial occlusion of the gut. These are anorexia, periods of vomiting, abdominal distension, borborygmus and colic. Any gastric symptoms present may be mistakingly attributed to the intestinal trouble. Symptoms may be referred to the rectum, or the rectum may be actually involved secondarily. Occasionally without the presence of discoverable growth there occurs hyperemia of the rectal mucosa with a distressing pruritus ani which is very difficult to relieve.

There are cases of gastric cancer marked by evidence of intestinal indigestion with fermentation and auto-intoxication. Although often there are borborygmi and signs of intestinal irritability, yet there may be constipation or irregularity of the bowels.

Certain cases of cancer of the stomach are accompanied by intense abdominal pain. This may be explained by extension of the growth beyond the stomach where it causes tension, or by metastasis to the spinal column. Osler reported a case in which severe neuralgia was occasioned by metastatic growth pressing upon the bronchial plexus.

By extension of the cancerous growth above the diaphragm thoracic symptoms are developed which mask the stomach symptoms. Pleurisy, empyema, lobular pneumonia and pericarditis, each may occur with its attending signs and symptoms.

A metastatic growth may compress the vena cava superior or inferior, or the subclavian vein, producing the well-known picture of local venous stasis. As a result of metastasis there may arise thrombosis and phlebitis in the veins of the extremities as well as of the trunk. Robert T. Morris reported cases involving the integument about the umbilicus and French observers have reported a number of cases in which the first evidence of the disease was

the appearance of growth at the umbilicus or in the umbilical vein.

Many other forms of latent cancer of the stomach might be described. Every large clinic provides such cases, some of them unique. Their occurrence teaches that gastric cancer is not infrequently the cause of symptoms remote from the primary disease, that the symptoms are misleading and that gastric cancer with metastasis should be considered whenever a case shows unusual symptoms, especially of the viscera.

## CHAPTER XV

# CANCER OF THE STOMACH: DIAGNOSIS AND TREATMENT

### DIAGNOSIS OF CANCER

The following points should be remembered as useful in detecting the onset of cancer:

- (1) The patient suffers more local distress than formerly and without apparent reason.
- (2) Motor symptoms, especially those of motor insufficiency, are aggravated, if already present, or appear if previously absent.
- (3) These motor and other new symptoms at first may be accompanied by no increase in the total acidity, and soon the free hydrochloric declines.
- (4) Upon decline in free mineral acid there usually appears lactic acid. At first this is found only occasionally and in small amount. It should be sought after a test meal free from lactic acid; the Boas test meal (oat-meal gruel) is given the night before the examination. Thorough lavage should precede the giving of the test meal.
- (5) The glycyltryptophan test is usually positive, as is also a strong antitryptic reaction in the blood and a hemolytic action of the blood serum.
  - (6) The presence of Oppler-Boas bacilli.
  - (7) A positive result from Salomon's test.
  - (8) Results of X-ray examination.

It is not to be expected that all of these will give witness early in every case. Yet they are our best criteria for diagnosis. There is great and often inexplicable variation in the behavior of cases. The symptoms at times could scarcely be overlooked; again, the transition from ulcer to cancer is so gradual as to deceive even after painstaking study.

Although failure of gastric secretion is justly accounted an important factor in the diagnosis of gastric cancer this feature must not be given too much emphasis, for commonly in old people there is low gastric secretion or anacidity without cancer. Gastric atrophy with loss of secretion in the aged has been noted by Ewald. Fenwick and others. Karjaard, who systematically studied this question, concluded that free HCl was often absent; Seidelin studied the gastric secretion in 70 old people and noted the absence of HCl in 14 per cent, and less than the normal standard in all excepting 10 per cent; Liefschutz, after the study of 60 cases, concluded that lowered secretion and even achylia gastrica was common in old people. Friedenwald 1 studied 27 cases over 50 years old, none presenting symptoms of gastric trouble, and found free HCl constantly absent in 44 per cent. It is to be concluded that undoubtedly there is an absence of gastric secretion in a considerable proportion of persons over fifty. The important bearing of this fact on the question of diagnosis of cancer of the stomach in old people is evident. It negatives to some extent the diagnostic importance of the absence of hydrochloric acid.

The symptoms of cancer when the disease is becoming advanced are cachexia, anemia, emaciation, pain, vomiting, hemorrhage and tumor. It must be admitted that any one of these may be absent. Sometimes the first warning of gastric cancer is a sudden onset of vomiting which persists, and is nearly uncontrollable. At other times hemorrhage, on rare occasions profuse, is the first

<sup>1</sup> Jour. Med. Assoc., 1908.

intimation of cancer. Pain is not often a constant sympton, and is so frequently entirely absent in this disease, though present in other affections, as to limit its diagnostic importance. Pernicious anemia, the chronic infections especially, syphilis, uremia and certain rare cases of chronic gastritis are the conditions most often confused with advanced cancer of the stomach.

Physical Examination.—Inspection frequently is valu-The patient should be placed upon a table of proper height; the light should fall obliquely from the direction of the patient's head; he should be requested to breathe slowly and deeply, when it may be noted if there is any peculiarity in the movement of the abdominal parietes in the region of the stomach. There may be seen, for instance, a slight prominence either fixed or moving with the deep respiratory efforts. If it changes position with inspiration and expiration, the tumor is probably in the pyloric region. When the tumor is located at the lesser curvature it does not move with the expiratory act, unless the stomach is adherent to surrounding organs in which case the expiratory immobility ceases to be a reliable guide. However, in such a case the tumor does not ascend during inspiration and, indeed, it may appear slightly to descend, as shown by Minkowsky. A tumor may become evident on distension of the stomach with gas or water though no sign of it could previously be seen. method is especially serviceable when the growth is located along the lower part of the greater curvature, or when it involves the anterior wall, or the pylorus. position of the tumor in relation to the topography of the abdomen may vary considerably with the emptying or filling of the stomach, especially in case of tumor of the pylorus. When the stomach is full, the mass may be seen or palpated rather high up and usually slightly to the right of the median line and when the stomach is empty, lower down and to the left of the median line. Tumors of the pylorus when not adherent to surrounding parts are movable on palpation. This is also, although less frequently, the case when the growth is located at any point of the lower third of the stomach.

In suspected cancer of the stomach, painstaking palpation should be made over all parts of the abdomen, by which means we may verify that which inspection led us merely to suspect. Attempt should be made to ascertain the position which the stomach occupies in relation to other organs in the abdomen. A very important diagnostic sign, much valued by Boas, is the occurrence of increased rigidity of the stomach. This may depend upon infiltration or over-tonicity of its walls. Inspection may cause this rigidity to be suspected, and its presence may be confirmed by palpation. It is sometimes necessary to procure as a provisional step the greatest possible relaxation of the walls of the abdomen. To begin with, very gentle stroking over the part from above downward should be practiced. For this purpose the tips of the fingers alone should be employed with a very light touch so that sensitiveness in the appreciation of resistance may be exercised to the fullest extent. By this measure deepseated resistance may be noted which escapes detection by other methods of palpation. This over-tonicity may be intermittent and when not palpable it may be excited and thus made evident by causing the patient to drink a glass of cold water. As the next step, deeper methods of palpation should be undertaken. At first palms of both hands should be used. The manipulation should be conducted so as to avoid exciting the contraction of the abdominal muscles. A growth in the stomach, at a point not covered over by the ribs, can be recognized almost invariably by these maneuvers. The contour assists in distinguishing a growth from a dilated gall-bladder. The liver should be carefully examined in view of the fact that metastases often occur in that organ. When the disease has attacked the liver the organ may be found enlarged with some irregularity of its surface. At times our suspicions of cancer of the stomach are first aroused by the rapid development of a secondary growth in the liver causing enlargement, deformity or both and sometimes producing jaundice or ascites. General edema is unusual except when cancer is far advanced and then it occurs as a part of the general cachexia. Dieulafoy confirms the statement of Trousseau as to the diagnostic importance of phlegmasia alba dolens. Local edema and umbilical phlebitis are signs to be remembered.

Robert T. Morris and T. S. Cullen have noted the metastatic invasion of the umbilicus in cancer of the stomach. Irregular enlargement of the pancreas, whether depending upon indurative pancreatitis or cyst, is sometimes mistaken for cancer of the stomach. Chronic enlargement of the head of the pancreas, causing obstruction of the common bile duct, presents a picture that sometimes resembles cancer of the stomach with metastasis in the liver. Cancer of the pancreas with induration and tenderness, but unaccompanied by jaundice may simulate cancer of the lower border of the stomach. There should be no difficulty in differentiation except in those cases in which cancer of the greater curvature or pylorus adheres to the pancreas. The pancreas is fixed during respiration and its indurated mass can be felt lying deep on the posterior wall of the abdomen. Usually the impact of the aortic pulsation is plainly to be felt transmitted through the tumor. This transmitted impulse is not unusual in gastric cancer.

In chronic pancreatitis the patient presents deterioration of general health, loss of weight, epigastric uneasiness and disagreeable dyspeptic symptoms. I have even known recurring vomiting to persist for years in these cases. Nevertheless, the recognition of chronic pancreatitis appears to me less difficult than is generally stated. I have repeatedly seen the diagnosis verified at autopsy and occasionally by surgical exploration. In cases that present elements of especial uncertainty, the colon should be emptied before palpation is attempted. Not often, but occasionally, a scybalous mass in the transverse colon may deceive the palpating hand.

Ptosis.—The stomach may be displaced and hence a tumor of the stomach may seem to spring from the intestine or the mesentery or to be in the retroperitoneal region. This is particularly true in the vertical stomach. When gastroptosis is associated with the vertical stomach, a tumor of the pylorus may be found as low as the umbilicus or even below that point. The lower border of the stomach may even be found in the pelvis. Of course the difficulty can be cleared up by using the gastrodiaphane or making a radiogram.

General Facts.—Less importance is attributed now than formerly to the occurrence of a large, left supraclavicular gland, sometimes called Virchow's gland, as a diagnostic criterion. Generally it is not present, and if found it may be but the result of cancer in other parts of the abdomen. Enlargement of the cervical, inguinal and other lymph nodes, is to be expected in old, slow-growing cancers. The French have called attention to enlargement of the lymphatics in the neighborhood of, and related to, the umbilicus. When cancer gives rise to ascites either from portal obstruction or from irritation of the peritoneum, the real nature of the primary trouble may be greatly

obscured. Likewise cancer of the stomach invading the thorax may be obscured especially if its manifestations are further masked by infection. Pleurisy, pleural effusion and abscess, gangrene of the lung, pneumothorax or pericarditis may develop from cancer of the stomach before the primary disease attracts attention. One case in my ward entered the hospital suffering from left pleurisy and pericarditis resulting from carcinoma primary in the fundus of the stomach. The stomach was perforated and adherent, yet abdominal symptoms were practically absent. The real nature of the trouble was not discovered until autopsy. A cancer which becomes infected with pathogenic organisms or which kindles a phlebitis may be accompanied by fever and other expressions of inflammation and may easily be mistaken for cholecystitis or other non-malignant troubles. Even without discoverable infection the course of cancer may be marked by intermittent or continued fever, and a high leucocyte count is not rare.

Cancer of the stomach, pursuing an unusual course may resemble obscure cases of ulcer, as in the following:

Case No. XXV. Cancer of the Stomach with Metastases to Lung and Liver Mistaken for Duodenal Ulcer and Cholelithiasis.—Nov. 13, 1912. Mrs. S., aged 50, Swedish, married, entered B. G. Hospital complaining of pain in the epigastrium and back. The family history was uncertain; her past history and habits seemed to have no bearing on her present illness. Six months before entrance the patient began to lose appetite; had pain in the region of the stomach. Then she improved slightly until about a month before entrance, when the pain became more severe and extended to the small of the back. The third week before entrance she vomited bright blood on three occasions, and much melena was evacuated.

On entrance the woman was anemic but not cachetic; weight 160 pounds; thorax, negative; vessels of neck pulsated unusually. The liver extended from the fifth to the ninth rib; there was a slightly tender area in the midline between the ensiform and umbilicus.

No cause for hemorrhage was discovered. The blood examination showed 20 per cent of hemoglobin, 7,500 leucocytes, 1712 erythrocytes; differential showed 62 per cent "polys," 24 per cent small lymphocytes, 14 per cent large lymphocytes, 0.1 per cent eosinophiles, 0.2 per cent basophiles. The stool was tarry in appearance, showed marked reaction to occult blood, but only a few erythrocytes by the microscope. The following morning an Ewald test meal was given and removed one hour later. Sixty c.c. returned, poorly digested, showing a total acidity of 11; free HCl, 2; combined chlorids, 3; organic acid and acid salts, 6; occult blood present; no lactic nor butyric acid; no bile nor albumose. Microscopic examination showed yeast cells, starch granules, but no special bacilli.

On Nov. 24 the hemoglobin was still 20 per cent (Sahli). At noon that day the patient was feeling comfortable and about to take dinner when suddenly she experienced intense stabbing pain located over the gall-bladder anteriorly, radiating to the back, accompanied by vomiting, clammy sweat, prostration and great pain.

On the 30th, X-ray examination by Dr. Plummer. Negative for calculus or disease of bone. The stomach was found small, lowest point opposite second lumbar vertebra; peristaltic wave shallow. Pylorus was patulous, its shadow continuous with that of the duodenum. The gut seemed fixed in a horizontal line. No evidence of tumor. The stomach and duodenum were emptied of bismuth after one and one-half hours. Plates suggested

fixation or narrowing of the pyloric end of the stomach and duodenum. On Dec. 9th there was a recurrence of pain such as occurred on Nov. 24th. Following this the woman steadily improved, although the stools and vomitus occasionally contained occult blood.

On Dec. 29th, hemoglobin was 42 per cent; Jan. 6th, 55 per cent, occult blood in the stools absent for the first time. Jan. 18th, hemoglobin 66 per cent, hemolysis was normal; antitryptic index normal; patient very well and was discharged from the hospital Jan. 18th. Diagnosis, probable duodenal ulcer, possibly also cholelithiasis.

After discharge the patient suffered somewhat from indigestion. On April 4th she had pain in the right axillary and mammary region with a feeling of tension. days later the pain became more severe and a physician was called. Patient had a profuse sweat, shortness of breath and pain on respiration: after three days pain subsided, then recurred with increased violence. tered the hospital on the 11th of April with normal temperature, pulse 110, respirations 30. There was evidence of fluid in the right pleural cavity. Blood examination showed hemoglobin 61 per cent; red cells, 4,730,000, leucocytes, 12,450; polynuclear, 76 per cent. The urine was negative; stomach contents not examined. The heart was displaced to the left, the liver two and a half inches below the costal margin. On the 13th of April the chest was aspirated, 1000 c.c. of very dark fluid was removed, specific gravity 1.018; microscopically many large cells apparently endothelial, besides blood cells in abundance. Aspiration gave only slight relief and dyspnea continued. There was little cough, no expectoration; on the 15th there was a recurrence of pain in the chest; the patient suddenly went into syncope and died.

The case was diagnosed as cancer of the stomach with

metastasis to the right lung and liver. There was no autopsy, therefore the original diagnosis could not be excluded. In favor of cholelithiasis were radiographic evidences of adhesions; in favor of duodenal ulcer was the prompt emptying of the stomach and duodenum, the abundant hemorrhage, the subsequent gain in blood and general health.

However, the metastasis to the lung, the hemorrhagic exudate in the pleural cavity and the manner of death makes it seem probable that the primary disease was cancer of the stomach.

Masked Cases.—Certain non-malignant affections sometimes strikingly resemble cancer of the stomach. As already stated, old and neglected cases of syphilis with cachexia may be accompanied by dyspeptic symptoms which are very misleading. The difficulty may be magnified when a gumma is discoverable in the region of the stomach. When any doubt occurs a Wassermann test should be made or a course of mercury and iodin tried. A more complete discussion of the differentiation between cancer and visceral syphilis will be found in the article on syphilis of the stomach. Tubercular peritonitis and visceral tuberculosis may be mistaken for gastric cancer.

Carcinoma may give rise to anemia closely resembling pernicious anemia and possibly might be mistaken for it. The points of resemblance are the lemon-colored tint of the skin, the high hemoglobin index, the small number of erythrocytes and the presence of erythroblasts. However, cancer causes greater emaciation than pernicious anemia. In cancer a high hemoglobin index is very exceptional as is the presence of numerous nucleated red cells. In pernicious anemia there is ordinarily a subnormal white count; according to Cabot below 5000 per c. mm. in 56 per cent of cases; and there is an absolute

decrease in the number of polynuclear cells while the lymphocytes remain near the normal point.

#### SPECIAL DIAGNOSIS OF CANCER

With the development of cancer there is a gradual and at times an abrupt disappearance of secretion of hydrochloric acid. In exceptional cases this does not occur until the disease is advanced. After the disappearance of HCl there are usually present the Oppler-Boas bacilli. When the growth occurs in the fundus of the stomach the rennin ferment may be low or absent; when the growth occurs near the pylorus, both pepsin and rennin are small in amount and the lessening of pepsin may be the more pronounced of the two. To this there are notable exceptions.

Before the macroscopic appearance of blood in the stomach contents, red cells may be found microscopically and chemical tests may show occult blood both in the contents from the fasting stomach and in the stools as first noted by Ewald and Kittner. The careful microscopic examination of the stomach contents may show red blood cells and leucocytes especially when there are disturbances of motion and secretion. Attempts are still made to discover nests of cancer cells in the stomach contents, and some enthusiastic investigators give the method unstinted approval. With most of us it has proved disappointing. Hemmeter recommended the curetting of the gastric mucosa by means of a stomach tube, having at its end a sharp edge, hoping thereby to disengage fragments of disease mucosa, which subsequently might be removed by aspiration. The plan has not met with general acceptance, and is open to important objections. Marini<sup>2</sup> reports his conviction, based on the

<sup>&</sup>lt;sup>2</sup> Arch. f. Verdg. Krank., Apr., 1909.

study of 37 cases, that the finding of tumor cells, even when isolated, points to the diagnosis of carcinoma and

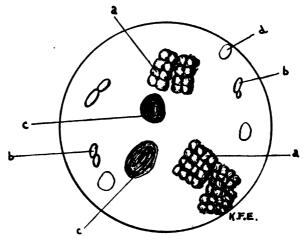


FIG. 35.—GASTRIC CONTENTS FROM A CASE OF BENIGN STENOSIS. a, Sarcinae; b, Yeast cells; c, Starch Granules; d, Oil globules.

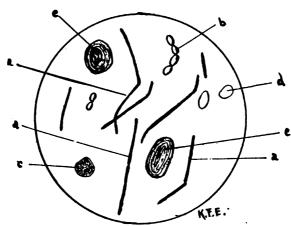


FIG. 36.—GASTRIC CONTENTS FROM A CASE OF CARCINOMA OF THE PYLORUS.

a, Long bacilli (Oppler-Boas bacilli); b, Yeast cells; c. Pus cells; d, Starch granules.

that these cells retain the characteristics of the epithelium from which they are derived, enabling one to form some

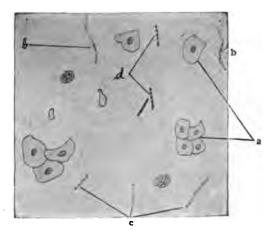


FIG. 37.3—LEPTOTHRIX FROM THE MOUTH IN A FRESH PREPARATION STAINED WITH LUGOL'S SOLUTION. (x350 Diam.) a, Squamous epithelium from the mouth; b, Threadlike leptothrix with distinct septa (without the iodin reaction); leptothrix gigantea Miller; c, threadlike leptothrix without septa and without the iodin reaction; d, "Long bacilli," giving a beautiful starch reaction.

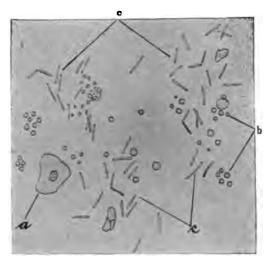


FIG. 38A.8—LONG BACILLI FROM STAGNATING ESOPHAGEAL CONTENTS, IN A FRESH PREPARATION STAINED WITH LUGOL'S SOLUTION, GIVING NO STARCH REACTION. (x350 Diam.) a, Esophageal (moral?) epithelium; b, Fat droplets; c, "Long bacilli," occurring abundantly.

<sup>2</sup> Figs. 37, 38A and B, 39, 40, 41, from Fricker's Article in Arch. f. Verdauungsk., XIV.

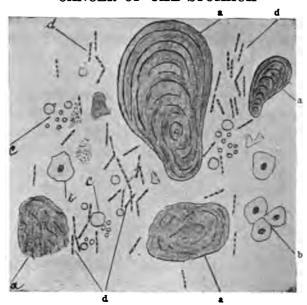


Fig. 38B.3—Long Bacilli from Stagnating Esophageal Contents, in a Fresh Preparation Stained with Lugol's Solution, Giving a Beautiful Starch Reaction. (x350 Diam.) a, Potato starch cells; b, Esophageal (oral?) epithelium; c, Fat droplets; d, "Long bacilli," occurring abundantly.

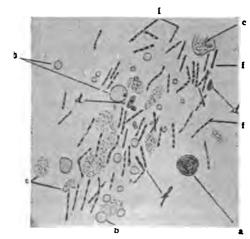


FIG. 39.3—LONG BACILLI FROM STOMACH CONTENTS IN A CASE OF GASTRIC CARCINOMA, IN A FRESH PREPARATION STAINED WITH LUGOL'S SOLUTION, GIVING A BEAUTIFUL STARCH REACTION. a, Cornstarch granules; b, Fat droplets; c, Flake of fat; d, Budding yeast; e, "Long bacilli," occurring abundantly.

idea as to the site of the tumor and the nature of the cancer. He believes that this is at times the earliest means of diagnosis. The work of Einhorn and a number of other earlier workers might be cited but it is enough to say that thus far the microscopic study of the stomach contents is of small value in the early recognition of cancer, excepting as it concerns the constant occurrence

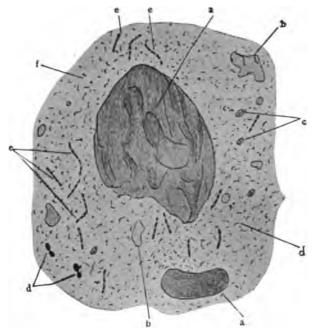


Fig. 40.3—Long Bacilli in Intestinal Contents in a Fresh Preparation Stained with Lugol's Solution, Giving a Beautiful Starch Reaction. a, Barley corn; b, Muscle fragment; c, Yeasts; d, Clostridium Butyric (Prazmowsky); e, "Long bacilli" occurring in considerable numbers; f, Intestinal bacteria and débris.

of blood and leucocytes which are often in tiny masses combined with bacteria and food detritus.

A good deal was hoped from the method of Salomon, which is as follows: The patient is given water freely in the morning, and at 2 P. M. food which is free from al-

bumin. At 9 p. m. the stomach is thoroughly cleansed by lavage and complete abstinence is required until morning. Then the stomach is washed with 400 c.c. of physiological salt solution, using the same solution twice. This solution is then tested by Esbach's and Kjeldahl's methods for total albumin and nitrogen. Salomon con-

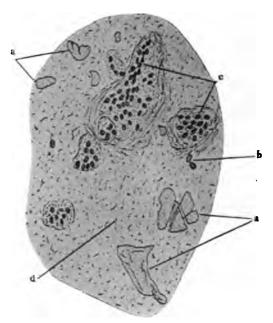


FIG. 41.8—CLOSTRIDIUM BUTYRIC (PRAZMOWSKI), IN INTESTINAL CONTENTS IN A FRESH PREPARATION, STAINED WITH LUGOL'S SOLUTION GIVING A BEAUTIFUL STARCH REACTION. (x350 Diam.) a, Muscle fragments; b, Yeast cells; c, Clostridium butyric on a starchy substratum; d, Intestinal bacteria and débris.

cluded that in all cases of carcinoma of the stomach Esbach's reagent produced a flocculent precipitate and Kjeldahl's method showed more than 25 mg. of nitrogen per 100 c.c. In most other stomach affections there was found only a slight cloudy precipitate with Esbach's, and not above 16 mg. nitrogen with Kjeldahl's method. Witte and others have endorsed this test but positive results

have been found in cases of simple chronic ulcer also. The reactions are believed to be absent in nervous dyspepsia, chronic gastritis, ptosis and in most ulcer cases. Richer found positive reactions in 4 cases of gastric cancer, 2 of achylia and 1 of subacidity. Goodman working with Musser,<sup>4</sup> after studying the method in 56 cases showed that the results were not uniform.

The Antitryptic Reaction and Hemolytic Action of Blood Serum.—The presence of isolysins in the blood serum of cancer patients, as described by Crile and Beebe, has been widely recognized, and an active hemolytic action may be regarded as characteristic of cancer. The absence of hemolysis does not exclude cancer; its presence in a marked degree does not warrant a diagnosis of cancer; yet, when tuberculosis is excluded, a marked hemolytic action of blood is of assistance in reaching a diagnosis of cancer.

The "cutaneous reaction" of cancer appears to be one manifestation of the action of the isolysins.

The antitryptic reaction in the blood serum, first announced by Marcus, is usually, but not always, present in cancer. In some of my cases, proved by operation or by autopsy to be carcinoma, the antitryptic reaction was absent. It may occur in non-malignant cases, in suppuration, in acute infections, etc. Nevertheless it affords a valuable indication. Out of 600 collected cases it was reported positive in nearly 75 per cent.

When suppuration, acute infection, etc., can be excluded and when a tumor and definite signs of local disease are discovered, then the presence or absence of the antitryptic reaction is of positive assistance in reaching a diagnosis.

Glycyltryptophan Test of Cancer.—The work of Fisch-

<sup>4</sup> Univ. of Penn. Med. Bull., May, 1909.

er and Neubauer, in 1909, attempting to identify a specific ferment in the secretion of the carcinomatous stomach, was suggested by the discovery several years before, by Müller and Lewin of a definite tryptic ferment in epitheliomatous tissue that would hydrolyse proteids and liberate amino acids. This ferment comported itself unlike ordinary gastric ferments but rather like pancreatic ferment.

It was of practical utility to use for the test some simple proteid which on hydrolysis would be resolved into end products that could be quickly recognized.

For this purpose they selected glycyltryptophan which, when incubated 24 hours in the presence of cancerous gastric juice at 38-40° C., liberated tryptophan, a substance easily recognized. Since the same result occurs in the presence of pancreatic ferment, to make the test of value, there must be assurance that duodenal contents has not adulterated the stomach contents by regurgitation through the pylorus. The test is therefore scarcely reliable when bile, doubtless carrying with it pancreatic secretion, is found in the stomach. It is stated that the preparation of glycyltryptophan deteriorates quickly and, to be reliable, the test must be made with a fresh specimen. Medina believes that the test is reliable regardless of where the cancer is located in the stomach, but he cautions that in highly acid contents the test is of no value.

It has been ascertained that the blood normally contains antitrypsin, a specific antibody that protects the organism from the action of the ferments that are absorbed. This antitrypsin increases during the digestive period, and is present in amount beyond the normal when, consecutive to obstruction of the duct of Wirsung, there is an unnatural absorption of pancreatic ferments.

These facts, according to Medina,<sup>5</sup> explain the unreliability of the glycyltryptophan test when blood is present in the stomach contents. The antitryptics in the blood nullify the special cancer ferment in the stomach contents.

Thus there has been shown a relation between the antitryptic blood reaction and the glycyltryptophan test of stomach contents. Used with essential precautions and in suitable cases, this laboratory method will probably grow in professional favor. Taken in connection with a strong antitryptic reaction and with a hemolytic action of the blood serum, the glycyltryptophan test seems to be of value for early diagosis of cancer of the stomach.

The work of Smithies 6 in Mayo's clinic confined to the glycyltryptophan test alone, indicates that a positive test is not pathognomonic. Used with stomach contents from which blood and duodenal fluid are strictly excluded, it becomes more reliable. The results are more significant when supported by the evidence of the antitryptic and the hemolytic tests than when considered alone.

Concerning the deviation of the complement, the presence of anaphylactic shock, the studies of the precipitins, the increased toxicity of the blood serum or of the gastric juice in cancer of the stomach—none of these have obtained a sufficiently reliable reputation to make them of practical value in the diagnosis of cancer of the stomach.

Albuminosuria, when appearing as a constant manifestation, is indicative of cancer. Aldor found it present in 50 per cent of cases and its presence has been recognized by many others.

In early cases the blood count is of little value. The absence of a digestive leucocytosis, to which Schneyer

<sup>&</sup>lt;sup>5</sup> Arch. des Mal. de l' Appar. Digest., Juin, 1912.

<sup>&</sup>lt;sup>6</sup> Arch. Int. Med., Oct., 1912.

called attention, is not a reliable indication as it may occur in other diseases besides carcinoma.

For the study of the bacterial flora in the stomach contents, Fricker has made one of the best contributions. There is found present a leptothrix called "the long bacillus," and other lactic-acid-forming bacilli. long branching organism, ordinarily called the Oppler-Boas bacillus, occurs in stagnating contents. may be found in other parts of the digestive tract than the stomach—in the mouth, esophagus or intestines, if conditions are favorable for its development in these locations. It occurs in the stomach especially when there is stasis, and when free hydrochloric acid is absent and, more rarely, when free HCl is present in moderate amount. Yeast and sarcinae grow in the stomach when there is a depression of motor function, but when hvdrochloric acid is present. The lactic acid bacilli and the Oppler-Boas bacilli require both stasis and diminution in hydrochloric acid. Since yeast and sarcinae occur in contents containing HCl they are rare in cases of malignancy of the stomach, but are present in pyloric stenosis from benign causes and in gastrectasis from atony. When long bacilli are present lactic acid is usually found also. Although the occurrence of these long and branching bacilli in the stomach contents may be expected in cancer of the stomach it cannot be considered as diagnostic. Taken in connection with the conditions already described, their presence helps to form the characteristic picture of cancer.

Phillip King Brown<sup>8</sup> speaks favorably of the method of Schmidt, of Nausser's clinic, for the study of the Oppler-Boas bacillus in the stools. The fact that this or-

<sup>&</sup>lt;sup>7</sup> Arch. f. Verdauugskr., Oct., 1809. <sup>8</sup> Jour. Am. Med. Assoc., Nov., 1909.

ganism is Gram-positive, serves to distinguish it from others that resemble it. Nausser states that the absence of Gram-positive organisms in the stools may exclude cancer. On the other hand their presence does not necessarily indicate cancer. I have not been able to satisfy myself as to this test, although Brown reports four cases in which the diagnosis was confirmed by autopsy or by operation.

Blood in Cancer.—Although the characteristic state of the blood in cancer of the stomach is that of secondary anemia, there are exceptional cases in which the blood picture may be deceptive. With marked gastric hypersecretion, with vomiting and diarrhea, the blood may become so concentrated that polycythemia occurs, and thus occasionally the red cells may rise above 6,000,000 per c. mm. A good description of the blood in this disease is found in the monograph by Osler and McCrae.9 They found that in 52 cases the average hemoglobin percentage was below 50, while the average number of corpuscles was 3.712.186. The erythrocytes exhibit the usual variations in size and shape found in secondary anemia. The white blood count is above the normal in about 50 per cent of cases. Occasionally there is a marked leucocytosis, for instance, 20,000 to 30,000. On the other hand there may be leucopenia, rarely below 4,000. Usually the polymorphonuclears are increased at the expense of the small mononuclear cells, while the large mononuclears are often relatively increased in number. As elsewhere stated cancer of the stomach may closely simulate pernicious anemia. In 129 cases Cabot found that in 26 the red count was below 3,000,000 and Emerson reports that in 16 out of 134 cases studied at the Johns Hopkins Clinic. the red count was equally decreased. In rare instances

<sup>9 &</sup>quot;Cancer of the Stomach," 1900.

it drops below this number, yet nucleated red cells are rare. Myelocytes are occasionally present, and in exceptional cases the condition of the blood very suggestive of pernicious anemia, which fact becomes control to the diagnostician if the other features of the case resemble that disease. Metastasis to the bone marrow, according to Rousseau, may lead to blood changes resembling those seen in leukemia.

The Roentgen Rays.—When cancer invades the gastric mucosa the structure of the surface is often loosened and irregularly softened so as to admit the entrance of small amounts of bismuth. In consequence the radiographic shadow at these points is blurred and uneven and lacks the definition observable in other regions.

Deformity of the stomach, protruding masses, persisting contractures, biloculations, etc., give rise to corresponding peculiarities in radiographic pictures.

Modifications of the normal peristaltic movements, the result of induration, ulceration, contraction or adhesion, are to be recognized in radiograms, though they are somewhat more satisfactorily demonstrated with the fluoroscope. (See Radiogram XIX.)

#### **PROGNOSIS**

The hopeless prognosis in cancer of the stomach not subjected to early operation need hardly be stated. Does cancer ever recede and make a spontaneous recovery? It would seem that in extremely rare cases this is true, although such an admission in the light of experience seems almost ridiculous. One of my cases, having a history that conformed with that of cancer of the stomach, developed cachexia, a palpable tumor, hematemesis and a characteristic gastric chemistry, yet the patient recovered, under palliative and symptomatic treatment. There was



Radiogram No. XV.—CARDIOSPASM.
Enormously dilated esophagus. Thirty years' duration, promptly relieved by stretching with Sippey's dilator.
Case seen with Dr. Eli Long.



Radiogram No. XVI.—ESOPHAGEAL STENOSIS WITH MODERATE DILATATION.



Radiogram No. XVII.—ESOPHAGEAL STENOSIS WITH DILATATION FROM CARCINOMATOUS INFILTRATION.



Radiogram No. XVIII.—ESOPHAGEAL DIVERTICULUM. Pulsion from ulceration and stenosis.



probably an error in diagnosis yet the facts were disconcerting. Similar cases have been reported.

### TREATMENT

Medical Treatment.—Medical treatment of cancer is not undertaken with expectation of doing more than procuring a greater degree of comfort and prolonging life. It has been said that there is no medical treatment for cancer and that surgery alone renders real service. This certainly overstates the matter. Most cases of gastric cancer require medical treatment even after operation. Experience has made me skeptical as to the ultimate cure of gastric cancer by surgery or other means except in a few most favorable cases. Great relief or respite of the disease may be procured, but rarely permanent cure. Ultimately, unless some intercurrent affection kills the patient, he returns to the physician for medical treatment, that is to say, for relief of his suffering.

It cannot be said that recurrence is invariable; occasionally there is a case in which the result of early excision has been so flattering that the skeptical look suspiciously at the microscopic section by means of which the diagnosis was verified. It is unfair to assume such a mental attitude as arbitrarily to hold in doubt rare cases of cure; such an attitude is not stimulating towards eager attempt at early diagnosis. The only enlightened course is that which retains hope for future diagnostic acumen of physicians and for elever work by surgeons. Among reports of recent surgical treatment, none exhibits greater calmness in statement, more careful discrimination, or more encouraging results than the contribution of Wm. J. Mayo. It dissipates somewhat the gloom that has so long surrounded this subject. The surgical re-

sults should excite efforts to reach that much desired "early diagnosis." To make this possible, no matter what future specific tests may be discovered, it will remain indispensable that all dyspeptic cases should be educated to report any noticeable change in general health or in local symptoms; each case should be carefully reviewed with diagnostic poise but with the possibility of cancer in mind.

The statement is frequently heard that all patients complaining of the stomach, who are not relieved by medical treatment in a given time (variously estimated from one to six weeks), should undergo surgical exploration. This arbitrary rule is unreasonable and highly improper until surgical expertness has rendered a celiotomy devoid of objection. To expect a physician to cure a serious disease within a definite time is to be oblivious to the natural history of pathological processes. No hard and fast rule applies; the exercise of sound judgment after all the evidence is obtained is the only conscientious guide. It is a mistake to assume that a surgical exploration necessarily lays bare the whole truth; in fact it often fails to disclose a cancer which is actually present yet inaccessible.

It would seem to be a safe rule to insist that all suspicious cases that do not promptly mend under medical treatment should be operated; that other cases, when the symptoms are against cancer and when they are reasonably accounted for, even when slow to improve, should be advised according to the diagnosis. Yet meantime the mind should be alert to note the beginning of the symptoms of cancer.

Surgical Treatment.—Surgical treatment, therefore, is urgently advised in all early cases of cancer with the hope that excision may for a long time delay and possibly cure

the disease. Even when the result is merely to postpone. the activities of cancer, operation is highly advisable. With cancer of the cardia or the lower end of the esophagus, little hope can be expected from an excision but an early gastrostomy permitting the comfortable feeding of the patient is an act of mercy. With cancer of the pylorus excision should be practiced when feasible, and when not, a gastro-enterostomy should be done. In the slowly growing cancers when the pylorus is obstructed, the results obtained by gastro-enterostomy are sometimes remarkable. In one of my patients where starvation had been carried nearly to the point of the patient's dissolution, there was a gain of forty pounds in the first forty days following an anastomosis by Dr. Park. The patient resumed her household duties, was able to walk several miles a day and became fully convinced that there was an error in diagnosis. After the lapse of a year she was apparently in good health; six months later, however, the disease had made great progress with metastasis in the liver, and the fatal termination came promptly.

Treatment of Vomiting.—Relief of vomiting may be obtained by temporary rest of the stomach, by lavage and other measures that soothe the diseased mucosa. The patient may be able to take nourishment on alternate days or two days out of three, providing he fasts strictly at other times. Lavage must be used with circumspection and with a definite purpose. Often it is wise to practice lavage at night, as an empty stomach may enable the patient to sleep and thereby prepare him to withstand the next day's ordeal. The use of bismuth after lavage, as in the cases of gastric ulcer, assists in relieving nausea and in quieting distress. Phenol in small doses, given in an emulsion, is useful in quieting vomiting and in relieving gastric unrest. Cocain, cerium

oxalate, codein or stovain may be combined with it. Some cases are resentful of every remedy and the vomiting continues until fasting is practiced.

Treatment of Pain.—Lavage does not succeed in relieving actual pain and in most instances should be avoided except to relieve distress occasioned by an overburdened stomach. When there is obstruction at the pylorus it may relieve pain temporarily and assist in obtaining rest at night. It is often a better plan merely to aspirate the contents without subsequently washing out the stomach. In using anodynes to relieve pain the least toxic should be chosen. Codein, the extract of jamaica, dogwood, cannabis indica, chloretone, chloroform water and the coal tar products should be tried before we resort to morphin. When the pain is severe, and when restlessness is intolerable, morphin should be given subcutaneously.

Treatment of Toxemia.—There frequently arises a distressing auto-intoxication, the result of secondary disturbances in the intestine and liver. Under these circumstances purgatives should be given and the colon irrigated. Although some drug that will excite intestinal peristalsis is indicated, a non-irritating purgative should be selected. The compound infusion of senna or cascara with magnesium carbonate may be tried. In some cases castor oil agrees remarkably well. It is a singular fact that in cancer the organism is very capricious as to the action of drugs; therefore considerable may be gained by making a careful selection. This applies not alone to purgatives and anodynes, but also to foods.

Treatment of Anorexia.—The disgust for aliment in gastric cancer is one of the most perplexing problems. The patient can do much to improve the appetite by exercising exquisite care in the toilet of the mouth and

DIET 457

teeth, which should be carefully cleansed with refreshing lotions each time after food is taken. Certain remedies may temporarily improve the appetite. Among these are the non-stimulating simple bitters, of which the infusion of condurango stands at the head. A freshly made infusion of gentian, quassia, or chirata is next in order. Occasionally the extract of nux vomica or strychnin are of real advantage, and I have observed a temporary response to the tannate of orexin. The patient at times craves pronounced flavors, and may be satisfied by the chewing of sassafras, calamus root, or by taking medicated waters or infusions. Important in this list are chloroform, camphor, peppermint, wintergreen or orange water. Putrefactive changes within the stomach may be corrected by giving a weak solution of chloral, in the strength of one part to 200 of water or by small doses of magnesium or bismuth salicylate. The ingenious practitioner may apply a great variety of changes, which result in relieving the symptoms and interesting the patient, which of itself is a feature well worth the trouble.

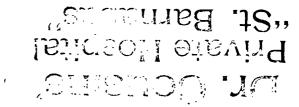
Diet.—If the patient is whimsical and capricious as to drugs, he is still more so in the matter of food, and the physician who approaches his case with a preconceived notion as to how alimentation shall be conducted is doomed to disappointment. Sometimes most unexpected articles of diet will agree better than those dictated by a sense of fitness. Perfectly fresh milk with the addition of a little lime water will be taken readily by some and abhorred by others. It is important to have milk that is not only fresh but pure, that is, having a low bacterial count. Occasionally milk becomes more tolerable when prepared as artificial buttermilk, or when converted into kiefer, matzoon or vito-lac, or when sour or curdled.

Some patients do well on peptonized milk and others take milk best mixed or boiled with barley water or infusion of other farina. Meat, fish and fowl are rarely liked and are usually detested. Occasionally broth or some of the meat extracts or powders prove serviceable. For a few days somatose or nutrose may be taken in considerable quantities. The salt smoked fish may be acceptable when uncooked although distasteful after cooking. fruits and fruit juices prove to be distressing. Sometimes orange juice is very acceptable as is a combination of fresh pineapple juice beaten with the white of egg. Among the various gruels I regard that prepared from the grated "flour-ball," 10 as the best. Of this a gruel or porridge may be made with milk or water or cream and water. Sugars and fats are usually badly tolerated, although some patients will accept light cocoa or an infusion of cocoa shells. Stimulants, especially alcoholic, nearly always occasion distress. Beer is an exception and is often not only tolerated but even enjoyed. During the periods when the stomach is left at rest, rectal alimentation should be employed and in its practice the rule laid down in the article on gastric ulcer may be followed. (See Page 340.)

Hygienic Care.—Much is to be gained by giving particular attention to the toilet. With the assistance of a deft and conscientious nurse comfort and rest may be given the patient through the prescribing of various local and general baths, such as brine, alcohol, cologne water or vinegar in proper dilution, thus stimulating the local nutrition of the skin. An oil or alcohol rub may so far allay nervous irritability that the use of anodynes may

<sup>&</sup>lt;sup>10</sup> Tie a pint of flour tightly in a napkin, throw it into a pot of boiling water and keep boiling steadily for five hours. When cold, the outer surface is removed and the interior grated into a fine powder.

be considerably curtailed. This is a marked advantage, as anodynes entail their own distress sometimes inducing discomfort more trying than actual pain, increasing the anorexia, constipation and biliousness, producing pruritus, depression of the urinary secretion and causing a loss of *morale* that renders existence nearly insupportable.



### CHAPTER XVI

## TUMORS OF THE STOMACH OTHER THAN CARCINOMATOUS

#### SARCOMA OF THE STOMACH

Gastric carcinomata usually develop from the glandular epithelium; sarcomata, more rarely met with, develop from the connective tissues of the submucosa, less often in the muscle layers, the lympathics or blood vessels. Sarcoma of the stomach has been considered rare, as dwelt upon by Dock in an excellent review of the subject; 1 however, some conclusions still more recent indicate that it represents about 5 per cent of malignant disease of the stomach. Its occurrence seems not to be influenced by sex, although somewhat by age, and it appears in the young more often than does carcinoma. though it may develop at any age, even at the extremes of life, it belongs to the first rather than the second half, in this respect differing from carcinoma.

Morbid Anatomy.—Sarcomata are divided into round cell, spindle cell and mixed types.

ROUND CELL SARCOMA.—The round cell type often shows extensive infiltration, and at times involves practically the entire stomach, leading to great thickening and encroaching upon the cavity of the organ. An infiltrating growth may involve especially one tunic of the stomach while other layers largely escape. Growing as it does from fibrous structure the disease is not, like carcinoma, primary in the gastric parenchyma. Even when the stom-

<sup>&</sup>lt;sup>1</sup> Trans. Assoc. Am. Phys., 1900.

ach is involved by a large growth the mucosa may not be affected; often, however, it is penetrated at one or more points, partly from the mechanical effects of the growth, partly from erosion. Occasionally the mucosa is widely destroyed.

SPINDLE CELL SARCOMA.—The spindle cell sarcomata, often described as fibrosarcomata, myosarcomata or angiosarcomata, more often appear as circumscribed tumors which grow from the periphery. They may start in the subserous fibrous tissues and then invade the contiguous parts so extensively that the point of origin is recognized with difficulty; they may have the appearance of extra-gastric growths. Beginning in the stomach, such tumors often involve the omentum, the pancreas or the surface of the liver. A sarcoma may take its origin in any region of the stomach; although it frequently begins at the pylorus, yet it is found almost as often along the greater curvature, the posterior surface or the lesser curvature, or infiltrating the stomach generally. Sarcoma is less often the source of obstruction at the cardia or pylorus than is carcinoma. The pyloric opening may be spared even when the growth is massive and greatly limits the capacity of the organ. To the naked eye the tumor may not be distinguishable from an epithelioma, a microscopic examination being necessary for differentiation. At other times the gross appearance is highly suggestive of sarcoma owing to the fact that the mucosa is slightly involved, while the walls of the stomach are greatly thickened from infiltration.

Sarcomatous growths may be single or multiple, primary or secondary. The round cell variety is especially accompanied by metastases particularly in contiguous organs. Slessinger<sup>2</sup> emphasizes the statement that

<sup>&</sup>lt;sup>2</sup> Zeit. f. Klin. Med., 1897, xxxii.

lymphosarcoma often leads to metastases of the skin. The same author notes that when the disease attacks the intestine, as it often does secondarily, it is not likely to lead to stenosis; whereas, in case of epithelioma, stenosis is the rule.

Clinical History.—The course of the disease has not been sufficiently studied and our knowledge of the symptomatology as compared with that of cancer of the stomach, is meager. It is known that the disease is often insidious in development; patients may suffer from marked cachexia and enfeeblement before the real nature of the disease is suspected. Some cases have been attended by high fever which has been mistakenly attributed to typhoid. At times the metastases, as for instance, those of the skin, have given the first suggestion of malignancy. Dock quotes a case of Baldy's in which there was no vomiting or other gastric symptoms; in fact the patient had a "voracious appetite." More commonly there is a loss of appetite, vomiting, hematemesis and gastralgia. When hemorrhage occurs it is a late symptom, owing to the fact that the mucosa is relatively undisturbed. The stomach contents in sarcoma closely resembles that of cancer of the stomach. Slessinger reported three observations, in the first of which there was absence of free HCl with abundance of lactic acid; in the second, a case of lymphosarcoma, there was a gradual transition from the normal secretion to anachlorhydria with the ultimate appearance of lactic acid and numerous Oppler-Boas bacilli; in the third case neither free HCl nor lactic acid was present. In each of these cases traces of blood were found in the stomach contents. Mathieu states that of . 17 cases there was total absence of HCl in 11.

Diagnosis.—Sarcoma of the stomach is usually not recognized save by histological examination of the diseased

structure; I owever, the ensuing facts are indicative of sarcoma; slow development, late involvement of the mucosa even when the growth is evidently far advanced, absence of stenosis when the disease attacks secondarily the intestine, metastases in the skin. Of the symptoms pain is the most frequent; vomiting is rather exceptional, and hemorrhage, when present, occurs late in the disease. The appetite is sometimes preserved to a remarkable degree. At times the fatal termination seems to depend upon the constitutional rather than upon the local effects of the disease although the onset of hemorrhage exceptionally may cause death before very great general disturbances take place.

Treatment.—Sarcoma yields better results to surgical measures than does carcinoma; fewer cases recur after resection and even when the condition precludes other operation than gastro-enterostomy the patients survive longer than do carcinomatous patients under like circumstances.

#### BENIGN TUMORS OF THE STOMACH

Non-malignant growths are important in part owing to disturbance in digestion which they sometimes occasion and in part because they may be mistaken for cancer. These statements might equally apply to certain foreign bodies in the stomach, such as gastroliths and hairballs. The myomata or lipomata may grow in the walls of the stomach, the former springing from the muscular layer, and the latter from the submucosa. Upon growth they project into the cavity of the stomach and sometimes are the cause of symptoms. A more common tumor is the polyp which may be fibrous or mucoid in character. The latter may be associated, according to Rokitansky, with chronic gastritis, the so-called polypoid gastritis. (See

Fig. 56, page 576.) The fibrous polyps may occur singly or in numbers, more often near the pylorus but sometimes near the cardia. They are occasionally found in the esophagus where their presence usually gives rise to dysphagia, although exceptionally the easy distension of the part permits the passage of food and even of the stomach tube in spite of the presence of a polyp of considerable size.

Adenomata and Polypi.—In the article on chronic gastritis and that on cancer of the stomach reference is made to mucoid and adenomatous degeneration of the glandular epithelium, which often assumes a polypoid form. These polyadenomata differ essentially from cancer not only in structure but in their clinical history; they develop in the mucosa, and while their appearance and the extent of growth varies in different cases, they do not penetrate the muscularis mucosae. These growths are of importance, first, because they occur more often than other benign tumors of the stomach and, second, because they are reputed to degenerate into carcinoma. are associated with chronic gastritis and also appear in connection with carcinoma and chronic ulcer. Although polyadenomata are essentially of one nature they are variously described in accordance with the forms which they assume. Several forms may be present in the same case. Sometimes there is a marked display of polyps, often having long pedicles, sometimes there is a diffuse adenomatous change occurring in plaques which occasionally involves almost the entire lining of the stomach. of considerable size may develop and undergo ulceration, or reaching large dimensions, they may interfere with the functions of the stomach, or, occasionally, being drawn into the cardia or pylorus, may cause obstruction. There are certain types of polyadenomata which, in different degrees, show a tendency to malignancy; some pathologists assert that it is not always possible to discriminate with absolute certainty between the benign and the malignant, that is to say, between the adenomata and adenocarcinomata.

Fibrous polyps may occur singly or in numbers, more often near the pylorus, but sometimes near the cardia. They are occasionally found in the esophagus when their presence usually gives rise to dysphagia, although exceptionally, the easy distension of the part permits the passage of food and even of the stomach tube, in spite of the presence of a polyp of considerable size.

Inflammatory masses are not rare and when located near the stomach often embarrass the diagnosis. (See Cancer of the Stomach, page 413.) They frequently develop at or near the pylorus, are usually secondary to peptic ulcer, either in the walls of the stomach, or in case of perigastritis, may be outside the organ. Syphilis is a notable cause of non-malignant growth that often simulates carcinoma. (See Syphilis of the Stomach, page 620.)

False Tumors.—At times a resisting mass may be palpated in the epigastrium that may be erroneously classed as a neoplasm. Such are the "phantom tumors" occasionally described. A common source of such induration is a local hardening or contraction in the muscles of the abdomen. The displaced left lobe of the liver and Reidel's lobe, in case of hepatic ptosis, may occasion a mistake. The same is true of displaced and very movable kidney or spleen. Cohnheim calls attention to the possibility of mistaking the pylorus for a growth, and shows that it may be detected by noting its relaxation, under the hand or by feeling it contract and harden when the patient swallows ice water. In gastroptosis the descending stomach leaves

above its lesser curve a region of the abdominal aorta unusually exposed. This prominence of the aorta is frequently mistaken for a tumor, especially when there is, or has been, a peri-aortitis. Einhorn has given an excellent description of "Apparent Tumors of the Abdomen," some of which were not easily explained.

Occasionally an epigastric hernia or an indurated pancreas may suggest a tumor of the stomach. Error of diagnosis may be avoided by resorting to distension of the stomach or colon, or by making radiograms, and by careful analysis of all the facts of the case.

Foreign Bodies.—Among the most common of the foreign bodies are the hairballs which occasionally are found in the psychopathic, following the habit of hairswallow-Gastroliths, more rare than enteroliths, are to be classed as pathological curiosities. It is unusual for any swallowed foreign body to be discoverable by palpation through the abdominal wall. I have made the attempt in a case from which subsequently knife blades, screws, etc., were removed by operation, yet I was unable to feel them through the parieties. Although large foreign bodies accidentally or purposely swallowed are sometimes passed in the stools, it is usually advisable, should gastric symptoms develop, to remove such articles by a gastrotomy. The symptoms produced are sometimes inconspicuous, especially in the mentally unsound and hysterical. the other hand, excitable people laboring under the mistaken belief that a foreign body has been swallowed suffer from distressing symptoms until the mistake has been corrected. My colleague, Dr. Roswell Park, had the experience on two occasions of meeting with patients who stated that they had swallowed a plate of false teeth.

<sup>3</sup> Med. Rec., Nov. 24, 1900.

Each patient complained of gastric distress and was conscious of the presence of the foreign body. However, on relieving the alarm and practicing a little delay the plate of teeth was discovered in each instance at the patient's home and not in the stomach, and the symptoms, of course, disappeared immediately.

Although foreign bodies have been ejected from the stomach as the result of an emetic such attempts are inadvisable. It is well to give agar-agar abundantly and to feed with coarse foods, so that the intestinal tract may become relatively distended. There is belief that the method lessens the danger of injury to the part and that it favors the discharge of a foreign body per viam naturalis.

Gastric Mycosis.—There are to be found in the stomach of some healthy individuals great numbers of saprophytic bacteria, yeast cells less often, and still more rarely molds. These organisms, while ordinarily inoffensive, occasionally become mildly pathogenic when there are at the same time present local chronic inflammation or cancerous ulceration. Areas of partly necrosed tissue have been found to be infiltrated with mycotic filaments recognized by staining with Gram's stain. This mycotic infection may be transmitted to the stomach from previous development in the pharynx. Cases have been reported which appear to have been primary in the stomach.

The endomyces albicans are very rarely pathogenic in the stomach, although in feeble infants and senile patients this organism may occasion lesions, which correspond with those produced in the mouth or esophagus in thrush or sprue. Small, elevated, whitish patches, at times umbilicated, single or confluent, may be found in the buccal or pharyngeal mucosa and, associated with these, may be found similar patches in the gastric mucosa, especially along the posterior wall.

The filaments of the mycelium may penetrate to the submucosa, causing redness and swelling, areas of the surface at times being covered with grayish-white pseudomembrane. This development of mycelium, while not of itself of importance, indicates cachexia or great lowering of resisting power in the patient, such as occur in the later stages of diabetes or other grave metabolic states.

Favus of the stomach has been reported in a few cases in which the disease is present on the surface of the body.

Actinomycosis of the Stomach.—Actinomycosis of the stomach is a rare disease which results from direct infection, or at times is caused by extension from the thorax. The disease leads to the formation of an abdominal tumor, not freely movable, hard, ultimately softening in places, and sometimes discharging externally. It is accompanied by fever, depression, pain and tenderness.

In a case reported by Mayo Robson,<sup>4</sup> after a period of emaciation, with epigastric pain, a tumor appeared below the umbilicus. It was hard, extensively adherent and upon incision discharged pus containing the characteristic organisms. Such tumors while hard, may have soft areas owing to suppuration: these may be palpable. Suppuration leads to sinuses through the tumor or along adhesions that may bind the tumor to adjacent organs or to the abdominal wall.

The symptoms are both local and general. In the former class may be mentioned tenderness, pain, vomiting and the presence of tumor. Among the general symptoms fever is important. It may be high and may be accompanied by emaciation and prostration. The temperature curve is that of sepsis. There are chronic cases

<sup>&</sup>lt;sup>4</sup> Surg., Gyn. and Obst., XIII, 1911.

in which the constitutional manifestations are less evident.

Prognosis.—Deep infection with the "ray fungus" is more fatal than when it attacks more accessible regions; however, recovery has resulted in a small proportion of cases.

TREATMENT.—The treatment indicated is incision, curetting of the ulcerated tissue, drainage and the administration of potassium iodid.

## CHAPTER XVII

#### HEMATEMESIS AND GASTRORRHAGIA

Hematemesis, meaning the vomiting of blood, should be interpreted differently from gastrorrhagia, or bleeding from the stomach. A person suffering from gastrorrhagia may or may not have hematemesis, and from the fact that blood is vomited one should not conclude that the bleeding has necessarily occurred in the stomach.

## HEMATEMESIS FROM BLEEDING OUTSIDE THE STOMACH

It is not an uncommon experience to meet with vomiting of blood in cases of epistaxis or hemoptysis, the blood having been swallowed; the same is true of hemorrhage from the esophagus when there is ulceration, varicosis, or aneurysm of small vessels. Aneurysm of the aorta, by producing erosion of the esophagus may lead to hemorrhage. At rare intervals we meet with bleeding ulcers in the lower part of the esophagus from which hemorrhage is sometimes very profuse. These ulcers may be peptic, syphilitic, carcinomatous, etc. A rare form is typhoid ulcer, which usually appears in the later weeks of The most common source of hemorrhage from the fever. the esophagus is found in varicosis of the esophageal veins, the direct result of increased portal blood pressure, usually due to hepatic obstruction particularly from cirrhosis of the liver. In some cases at autopsy a minute perforation of the vein will be found, probably the result of erosion or trauma caused by the swallowing of some

hard substance or the passage of a sound. The extent of hemorrhage from a minute lesion is remarkable. The mistake is not infrequently made of attributing such hemorrhages to gastric ulcer. In my experience, the hemorrhage is difficult to control, and from it I have seen more fatalities than from hematemesis of any other cause.

## HEMATEMESIS FROM BLEEDING WITHIN THE STOMACH. GASTRORRHAGIA

When it is determined that the bleeding occurs in the stomach, the mind naturally turns either to gastric ulcer or cancer as an explanation; but while first, ulcer and second, cancer, are commonly the cause of gastric hemorrhage, these by no means account for its occurrence in all cases. Hemorrhage from the stomach may be produced as the result of hepatic cirrhosis, or other form of portal obstruction, in a manner analogous to hemorrhage from the esophagus already described. It may be associated with varicosity of the stomach vessels, or varicose vessels unrelated to hepatic disease may cause gastric hemorrhage. It may result from minute erosions of the gastric mucosa, and occasionally the bleeding is profuse when no actual lesion of the gastric mucous membrane can be found. In such cases the stomach seems unusually hyperemic, and its mucous coat has a turgid appearance and bleeds with the slightest handling. In the search for bleeding ulcer, which was not discovered, I saw the lining of the stomach bleed at many minute points from which blood oozed in considerable quantity even though the utmost care was practiced by the surgeon in examining the lining of the stomach inch by inch. The gastrotomy was performed with the intention of exsecting a supposed ulcer for the relief of long-continued hemorrhage. (See Case No.XVIII, page 310.)

Bleeding from the gastric mucosa, in the absence of ascertainable lesions, is discussed by Ewald <sup>1</sup> under the title "Parenchymatous Gastric Hemorrhage." He recites a case, that of a man aged 24, in which the bleeding proved fatal, yet at autopsy no cause was found for the hemorrhage, either in the stomach or elsewhere. In other cases described by Ewald gastrorrhagia without discoverable lesions of the mucosa resulted from vicarious menstruation, from obstruction in portal or splenic veins or from polycythemia.

Gastric hemorrhage may follow a congestion of the abdominal viscera as a part of the general stasis from cardiac failure or from pulmonary disease. Ewald calls attention to the fact that gastric hemorrhage may occur as the result of marked circulatory disturbance in the intestinal vessels such as follows invagination or strangulation.

Occasionally we meet with ecchymosis of the gastric mucosa as a part of systemic disturbance in general cachexia; profuse hematemesis may then occur as, for instance in scurvy and purpura and especially in giant purpura.

In one instance of the latter the hematemesis and melena were so extensive and continuous that I was led to despair of the patient's recovery, yet after a long illness improvement began and there was a happy issue of the case. Such a termination is unusual. In late syphilis, uremia, splenic anemia, leukemia, some types of malaria and other diseases characterized by degeneration of the blood, hemorrhage into the alimentary tract and often fatal bleeding are by no means rare. Many times very little structural change can be found at autopsy to explain this serious hemorrhage. It is to be accounted

<sup>&</sup>lt;sup>1</sup> Buffalo Med. Jour., May, 1913.

for by a loss of coagulability of the blood, at times depending upon a low calcium content.

Hematemesis is not rarely a feature of erythremia or primary absolute polycythemia, a condition of peculiar cyanosis first recognized by Vaquet and often spoken of as Vaquet's disease.

Jacobson and Post <sup>2</sup> have drawn attention to the occurrence of gastric and intestinal hemorrhage as an early result of severe infection and toxemia.

Some curious instances of hemorrhage apparently result from vasomotor disease. In one of my cases, that of a neurotic woman, there occurred repeated attacks of gastrorrhagia with vomiting of blood, sometimes but not always occurring at the menstrual epoch. This patient had, appearing upon her breast, neck and arms from time to time, large hyperemic areas from which the blood sometimes escaped freely. These vascular dilatations would appear suddenly, the skin on the preceding day having a normal appearance. They sometimes disappeared quickly, leaving a certain amount of discoloration or, again, continued for days, sometimes for weeks. In spite of this hemorrhagic tendency, the woman pursued her calling as a washerwoman with little interruption to her work. She eventually became tuberculous, but the advent of this seemed not to produce any diminution of her strength or her hemorrhages.

The vomiting of blood in lieu of the catamenial discharge I am satisfied is a reality. Undoubtedly this conception of hematemesis is often a mistaken one, and such explanation of the bleeding should be accepted only after most careful exclusion of other causes. When that is done, however, there will remain a few cases which seem not susceptible of other explanation.

<sup>&</sup>lt;sup>2</sup> Am. Jour. Med. Sci., March, 1912.

### 474 HEMATEMESIS AND GASTRORRHAGIA

In one of my cases, a neurotic girl of 18, who suffered from amenorrhoea there was periodical hematemesis for years. Venesection practiced on the first appearance of symptoms had the effect of stopping the vomiting of blood and ultimately the catamenia appeared with fair regularity.

Bleeding from Gastric Ulcer.—There is no agreement among observers as to the frequency of hemorrhage in This matter has been studied in cases of gastric ulcer. many clinics and the conclusion is reached that in something like 50 per cent of all cases hemorrhage may be demonstrated. It is probable that bleeding in a microscopic sense occurs in nearly all cases, but necessarily a percentage of these escape detection even when carefully studied. It may be assumed that with the frequent practice of lavage, examining the stomach contents as well as the feces, occult blood at least should be discovered in the great majority of cases. Clinically, microscopic hemorrhage should not be viewed as gastrorrhagia. It is true that occult bleeding may through carelessness go undiscovered, even if sufficiently important to produce serious anemia, but this would be impossible in cases properly investigated. In a clinical sense the term "gastrorrhagia" has reference to the escape of blood from the stomach in sufficient amount to be recognized macroscopically either in the stomach contents or feces. tent of the hemorrhage depends for the most part upon the location of the ulcer and the size of the artery opened by it. Fortunately large vessels often escape. When the coronary, the pyloric or the gastric branch of the hepatic or splenic arteries are eroded, the hemorrhage is terrific and is usually beyond control of the physician. considerable escape of blood may occur around the base of an ulcer, and this is sometimes aggravated by the ten-

sion of the stomach walls, which varies just enough to prevent the formation of an occluding thrombus. times this thrombus may be found in a vessel with blood escaping around it. In a series of 31 cases of ulcer producing stenosis at the pylorus, I could procure a history of vomiting of blood in but five, yet many of these were old cases. While bleeding of greater or less amount is to be expected in cancer, the actual vomiting of blood is not of frequent occurrence. In a series of 21 consecutive cases of pyloric cancer recently studied. I found that hematemesis had occurred in 5, but in none was the hemorrhage large in amount. Hematemesis occasionally is produced by gastritis, both acute and chronic, although this is an exceptional event occurring generally in those cases that have followed upon long continued congestion sometimes from portal, sometimes from general venous distension.

A number of instances of gastric hemorrhage in arterioclerosis have recently been reported in which bleeding without the intervention of ulcer came directly from degeneration of the arterial walls.

No lesion of the mucosa was found in these cases, save ecchymosis and minute separation of superficial tissue, dependent upon ruptured miliary aneurysm.

The Symptoms of Gastrorrhagia.—The patient usually suffers no pain during the progress of the hemorrhage. Moreover, pain previously present, even when severe, generally disappears with the oncoming of the bleeding. A sensation of unusual warmth in the epigastrium is experienced and a feeling of languor, later of faintness with palpitation of the heart, yawning, nausea, coldness of the extremities, sweating and the usual phenomena of hemorrhage. Nausea sometimes appears at once even when the loss of blood is slight, at other times even with profuse

hemorrhage, not until blood has remained in the stomach for some time. As a rule, when the bleeding is scanty, no symptoms are recognized. When blood in large quantities is vomited, it may have a varied appearance. depending upon the duration of time that it has remained in the stomach, the activity of the gastric secretion and the character of the ingesta. In case of actively bleeding ulcer one may expect to find some red blood, together with dark coagula and blood changed by the action of the gastric juice; when so changed it is a dark fluid, somewhat granular in character, the well-known "coffee grounds vomit." When such blood is poured into a glass, a dark sediment resembling coffee grounds collects at the bottom and the supernatant fluid has a brownish appearance. The appearance of the vomitus is of course modified by the presence of the more or less undigested food mixed with it and by the coloring matter that may have escaped from the food. Some amusing mistakes arise when patients discover an unusual color in the vomitus which. following the taking of certain fruits, wines, etc., suggests to the unpracticed eye the presence of blood. Several times I have hurried to some patient who had become unnecessarily alarmed at the appearance of vomited matter which actually contained no trace of blood. The bleeding from a cancer although rarely large in amount, is often very continuous. The vomitus is usually dark in color, may have the coffee grounds appearance and there are commonly present mucus and fragments of food. In most forms of gastric hemorrhage, except when extensive, the loss of blood ceases with careful emptying of the stomach by the skillful application of lavage with ice water, but this is less often true of hemorrhage from a cancerous The very fact of continuous oozing with the presence of mucus is strongly suggestive, if not diagnostic,

of cancer. In severe gastric hemorrhage, the stools are nearly black in color, the red blood cells may be for the most part broken down through the action of the digestive ferments. The appearance of the melena is characteristic, but to the inexperienced eye the evacuations following the taking of charcoal, bismuth or foods rich in pigment may be mistaken for melena. croscopic examination of the stools, while important, is not sufficient; the examination by chemical tests is required. When a differential diagnosis is sought in carcinoma the test for occult blood should be made daily. is almost uniformly present for the reason that oozing is relatively continuous. On the other hand, in ulcer the flow of blood is often interrupted or intermittent. finding of blood on one occasion and not on another is presumable but not conclusive evidence against cancer and it should be remembered that certain chronic ulcers bleed for a long time and at periods almost continuously. The importance of examination for occult blood is preeminent, and a conclusion should not be reached without long repeated and painstaking investigations. There is no place for casual observations. In cases where neither hematemesis nor melena are discovered, inconspicuous hemorrhage may be sufficient to produce anemia that is highly suggestive. It is not uncommon to have patients apply for relief of languor, faintness and other symptoms of anemia so characteristic that the loss of blood is at once suspected. However, the diagnosis is not always readily made. If the anemia depends upon the escape of blood from chronic ulcer, the hemorrhage may be so intermittent that only the zealous physician will discover blood in the stools.

The symptomatology of hemorrhage is so well known that one would expect that all serious cases could be

easily recognized. This, however, seems not to be the case.

The occurrence of gastric hemorrhage without premonition may show itself by syncope, probably the result of the sudden loss of blood. Such attacks may be the first intimation of the presence of the disease upon which it depends. When syncope occurs without the vomiting of blood the real condition of affairs is sometimes overlooked. Many years ago I had an experience which illustrates this. I was asked in consultation to see a young girl who having been exposed to scarlatina was supposed to be suffering from a severe onset of that disease. There was alarming pallor, the pupils were widely dilated, and only a flutter of pulse could be felt. She was approaching unconsciousness and had a sighing respiration. Nothing positive was learned by physical examination, except that the epigastric region was distended. There had been no vomiting nor pain. She had the classical symptoms of internal hemorrhage, and it was concluded that she was bleeding from the stomach and therefore hematemesis was predicted. While we were explaining the situation to the friends the girl vomited an enormous quantity of much clotted blood and then lapsed into complete unconsciousness. I did not see the patient again, but learned that she died on the following day. Although such cases are rare it should be remembered that they do occur. More often vomiting accompanies a profuse gastrorrhagia, and Ewald thinks that blood within the stomach acts as an emetic. This is probably true, but it should be remembered that vomiting and other spasmodic reactions are normal accompaniments of profuse hemorrhage from any source and that nausea and vomiting may result from syncope no matter what the cause.

Treatment.—The measures to be adopted in case of

hemorrhage from gastric ulcer and cancer respectively are described in the chapters devoted to those subjects. Briefly they include, in the case of ulcer, complete physiologic rest, to secure which morphin and atropin subcutaneously may be required besides the local use of adrenalin and ice water. In the case of cancer, physical and physiologic rest usually suffice. Consideration should be given to the state of the blood especially as to its coagulability. Impaired coagulating power often depends upon deficiency in calcium and benefit promptly follows its introduction. In gastric hemorrhage, in order that the composure of the stomach may not be disturbed, calcium lactate should be administered in the form of a clyster, 1 or 2 gm. dissolved in 250 c.c. of normal saline solution. This may be repeated three times a day for a period of three or four days and then discontinued. Subsequently if the hemorrhage has disappeared or is but slight, the drug may be given as a preventive measure two or three successive days of each week. Although our experience with the sera in the control of hemorrhage is not of long duration, we apparently have in this method a real acquisition. All foreign sera seem to be efficient, but it is desirable in their use to avoid anaphylaxis, and therefore certain sera are preferable to others. In an emergency diphtheria antitoxin, or the anti-streptococcus serum may be used, but of course it is better to employ the simple horse serum. Rabbit serum is less often attended with unpleasant reactions, and in Buffalo we have had good results from the sterile dried horse serum prepared according to the method of Busch and Clowes, and marketed under the trade name "Coagulose."

Ergot still remains the favorite remedy with many and is really efficient. It is best given in the form of ergotin hypodermically. Stypticin, hydrastin hydrochlorate, the fluid extract of hydrastis and hamamelis and emetin are valuable agents especially in small hemorrhages. Gelatin solution is not to be disregarded. We are not yet fully acquainted with the action of adrenalin in the treatment of hemorrhage. It overcomes tonus in the stomach and intestine; it is most effectual by local application.

Wiggers of Detroit from experimental work <sup>3</sup> concludes that large doses of adrenalin cause a short preliminary increase in hemorrhage and that on that account these should be avoided. Small doses cause little or no preliminary increase, shorten the course of the hemorrhage and are therapeutically desirable. Little result is obtained by subcutaneous administration, but by continuous intravenous injections of weak solutions slight elevation of pressure can be maintained and hemorrhage simultaneously checked. A good result may be accomplished by intramuscular injections. The state of the blood pressure is the criterion for the use of adrenalin in brief hemorrhage. With only slight fall in blood pressure the nitrites prove more beneficial than adrenalin. When the bleeding has been profuse, however, and a low pressure exists, it becomes vital that the hemorrhage should be checked without further reduction of pressure and adrenalin is strongly indicated. The administration of adrenalin should always be closely followed by observation of blood pressure. The smallest dose that is competent to raise the blood pressure is that which should be selected.

<sup>&</sup>lt;sup>8</sup> Arch. for Int. Med., March, 1910.

### CHAPTER XVIII

#### GASTROSUCCORRHEA

In 1882 Reichmann described a case presenting a group of symptoms which came to be regarded as a clinical entity and to which he gave the name gastrosuccorrhea. Essentially the disease depends upon an excessive secretion of acid gastric juice, and the presence of this secretion within the stomach at a time when the stomach should be empty. When this over-secretion is persistent it is called gastrosuccorrhea continua. In some cases the over-secretion is present merely for a few weeks, after which the patient is without trouble for an indefinite period and then the over-secretion returns. spoken of as gastrosuccorrhea periodica. Not long ago. Strauss described a group of cases in which the excessive secretion occurs only with the taking of food. fasting the patient escapes the over-secretion, but directly upon taking food, the excessive flow begins. this condition the name "digestive gastrosuccorrhea" has been applied.

## GASTROSUCCORRHEA CONTINUA. PERSISTENT SE-CRETION OF GASTRIC JUICE (RIEGEL)

The earlier reported cases of gastrosuccorrhea belong to the chronic or continuous form. Reichmann and most other contemporary observers unhesitatingly ascribed the trouble to a definite functional derangement of secretion. Riegel and others declined to regard it as a distinct disease, but looked upon it merely as a perversion of secretion due to a variety of causes. Nevertheless, for the most part it was held to be a definite neurosis. The more detailed study of cases showed that the continuous flow of gastric juice was often associated with other diseases, and Riegel came to the conclusion that it might result from any form of chronic irritation of the gastric mucosa. A similar conclusion was made by other observers who attributed the derangement to irritation from gastritis, to gastric ulcer and pyloric stenosis. It is not easy to disprove this view of the etiology of gastrosuccorrhea continua, but it must be admitted that there is occasionally a case which cannot be accounted for by any accompanying disease and in which the symptoms seem to exist as a morbid entity. Emphatic differences of opinion have been expressed on this subject.

Symptoms.—The symptoms of gastrosuccorrhea are fairly definite. (1) There is epigastric pain appearing about four or five hours after a meal; (2) there is found in the stomach before breakfast a large amount of gastric juice; (3) this gastric juice has usually a high degree of acidity, depending upon free hydrochloric acid: (4) the digestion of proteids is rapid while that of starches is usually delayed. To these cardinal symptoms a number of others of less importance must be added. The patient has usually a good appetite, an unusual thirst and a feeling of distension before the meal is completed. With some patients the sense of distension is more or less constant. Clapotage is easily developed, even while the patient is fasting, and the lower border of the stomach is generally found to be several centimeters below its usual position. I have never determined this question by means of the X-ray, but there can be little doubt that moderate dilatation frequently accompanies the disease. Gastric atony occasionally develops, while in other cases

there is excessive motor activity. Morning vomiting of pure gastric juice is persistent in some cases. The patient is temporarily relieved of his symptoms by lavage. Early in the morning one may withdraw several hundred c.c. of pure gastric juice of high acidity which, when turned into a glass, is found to arrange itself in three layers: the upper one light and foamy; the middle one somewhat opalescent, the lower, marked by flocculi containing starch detritus and flakes of mucus. Ordinarily this fluid gives a markedly positive starch reaction, and when heated it develops gas through fermentation of carbohydrate. A patient suffering from gastrosuccorrhea is generally constipated and may complain of great nervous irritability and headache. I have known this to occur in instances in which the secretion, though continued, was not very excessive, cases in which one would find not more than 50 or 100 c.c. in the fasting stomach. Yet the relief experienced by lavage was striking; and this refers not alone to the stomach symptoms, but to other subjective conditions. Severe cases of gastrosuccorrhea are infrequent. Many cases of moderate intensity escape notice, being classed as unusually rebellious instances of hyperchlorhydria. It is well to be alert in the examination of those cases of hyperchlorhydria complaining of symptoms while fasting.

Prognosis.—Continuous gastric secretion usually depends upon some concurrent disease, and recovery will depend upon the successful treatment of this exciting cause. In those rare instances in which there exists no local irritation of the gastric mucosa, and no obstruction, Reichmann's disease is very intractable to treatment.

# GASTROSUCCORRHEA PERIODICA. GASTROXYNSIS (ROSSBACH)

Whatever may be thought of the possibility of continu-

ous gastric secretion as the expression of a nervous perversion. I think there can be no doubt as to the validity of temporary nervous over-secretion. It is often observed in hysterical and neurotic individuals and is found to appear at times of great nervous strain. If there is any accompanying gastric disease, it usually escapes detection. The symptoms of gastric distress duplicate closely those occurring in continuous secretion. may be added that in these transient cases the symptoms are more acute and the accompanying gastralgia, headache and vomiting are sometimes distracting in character. These cases are commonly regarded as the expression of prolonged sick headache or migraine of unusual severity. The condition has been attributed to eyestrain, to underlying gouty conditions and to the epileptic equivalent. It may parallel closely the gastric crisis of tabes. (See Gastric Syphilis.)

That gastric secretion can be augmented by psychic or other nervous excitement is well established. Bogen noticed that an increase occurred as the result of a variety of psychic or associated stimuli even when the patient was fasting; for instance in one it occurred after the blowing of a trumpet. According to Bickel this view is substantiated by experimental work. On the other hand, it is conceded that intermittent hypersecretion may occur merely as a symptom of motor insufficiency, as shown by Forschback and others, or it may result from acid gastritis or irritative catarrh, sometimes with and sometimes without hyperacidity. It has been recognized as a temporary complication of gastric ulcer in which motor insufficiency usually plays a part. When there occurs a weakening of the muscle coat of the stomach, from degeneration or from nervous causes (gastric atony), one will occasionally find temporary hypersecretion.

To summarize, gastrosuccorrhea periodica may be but a symptom of local disease of the stomach; it may result from definite disease extrinsic to the stomach, as for instance, irritation at the nucleus; or finally, it may occur as a purely functional disturbance, analogous to nervous over-salivation, a condition that may accompany it. This association with sialorrhea may at times be something more than accidental.

Case No. XXVI. Gastrosuccorrhea.—In one interesting case in which there was regurgitation of gastric juice, eructations of gas and constant distress, and in which several ounces of gastric juice containing food particles could be withdrawn from the fasting stomach, the patient at the same time suffered from leucoplakia. The case improved after the practice of lavage and the administration of bismuth and light magnesium in full doses. In this case the sialorrhea doubtless resulted from the leucoplakia. It may be added that the patient's blood gave a positive Wassermann reaction.

#### DIGESTIVE GASTROSUCCORRHEA

Strauss first described a type of hypersecretion that appears after the ingestion of food but is not present in the fasting stomach. As it occurs only in association with gastric digestion, it differs from other types of gastrosuccorrhea periodica. In the latter affection there are times in which over-secretion exists whether the patient is fasting or not. Undoubtedly there has been some confusion between periodic and digestive gastrosuccorrhea.

Julius Friedenwald has recently reviewed the subject and pointed out the various aspects of the condition as seen by different workers. The essential particularities are: (1) the appearance of the excessive secretions in association with gastric digestion; (2) the presence of the symptoms of hyperacidity, although the actual acidity is not above the usual standard; (3) there is commonly a greater loss of weight than would be expected in view of the amount of food taken.

In addition to these three striking features there are observed the symptoms belonging to other types of oversecretion, for that matter, to other gastric derangements, for instance, marked constipation, various sensory or motor symptoms, such as pressure, fullness, "goneness," burning or, at times, pain; there may be eructations of gas, a feeling of contraction of the stomach, or actual atony may develop. Vomiting may be conspicuous or absent.

Diagnosis.—Gastrosuccorrhea may be recognized by giving the patient as a test meal, dry bread or cracker without water. Forty-five minutes later the stomach contents should be withdrawn by aspiration. At this period of digestion the normal stomach would contain very little. Any contents present would have a consistency so great that its passage through the tube would be difficult. the case of hypersecretion there is obtained a liquid content of 100 or 200 c.c. or even more. The average specific gravity of normal stomach contents after an Ewald-Boas test meal, removed after the lapse of an hour, is from 1.015 to 1.020; whereas in hypersecretion the specific gravity is 1.012, 1.010 or even lower. Thus it will be seen that the presence of a large amount of fluid contents, having an abnormally low specific gravity after a dry test meal is diagnostic of digestive gastrosuccorrhea.

Treatment.—For the permanent relief of over-secretion we should attempt the removal of the underlying cause. More immediate measures consist in early morning lavage, the administration of large doses of bismuth, the

hypodermic injection of atropin and the control of nervous excitability. Relief may follow a sojourn in the mountains or at the sea; but the trouble recurs when these patients resume their vocation, especially when it involves mental worry and responsibility.

#### THE EFFECT OF EYE-STRAIN

The prolonged use of the eyes in writing, reading or looking too intently and continuously at distant points should be avoided and errors of refraction should be corrected. I have seen gastrosuccorrhea develop in astigmatic locomotive engineers. It is more prone to occur at the age when visual accommodation is losing its efficiency. In such cases relief often attends a careful correction of refraction. This is not universally true, for the trouble with the locomotive engineer lies partly in the fact that he is looking intently at objects in a rapidly changing field: the accommodation is repeatedly and suddenly strained through the jarring and oscillation of the engine. This produces an intense aggravation of the ocular apparatus, and many times causes a sympathetic nervous disturbance of considerable importance. Every traveler in rapidly moving vehicles has experienced in a lesser degree the eve-strain to which I refer. This results in the familiar car-sickness. Even sea-sickness may be to a considerable extent avoided by the wearing of properly fitted eye-glasses. A proportion of these sufferers from car or sea-sickness at the time of the uncomfortable experience have over-secretion and may vomit considerable quantities of thin acid, watery mucus which is little else than gastric juice.

#### CHAPTER XIX

#### MOTOR INSUFFICIENCY—GASTRIC ATONY

In the chapter on gastric neuroses consideration is given to gastric atony. The subject extends beyond the domain of the nervous system and requires a rather wide view, especially in relation to motor insufficiency. thenia gastrica depends for its origin sometimes upon congenital and sometimes upon acquired conditions. congenital cases the weakness of tissues is not often lim-· ited to those of the stomach, but may be widespread and the stomach may be defective only along with the other muscles of the body. Individuals with this defect of development show an attenuation of muscles and a laxity of connective tissues. There is usually a flabbiness of the skin and, owing to this deficient tonicity, there will be found a pendency of the tissues of the mammae, buttock, abdomen, etc. Such individuals have feeble respiratory movements, intestinal inactivity and debility of the genito-urinary apparatus. In women there is apt to be some degree of uterine and ovarian prolapsus and in men the development of hernia and varices. Such persons have weak mesenteric ligaments and suffer from enteroptosis. Similar conditions are acquired from long continued malnutrition, anemia and physical inactivity, and are seen to result from the chronic infections, such as tuberculosis and syphilis, or they may follow grave illnesses like typhoid fever.

Shutz announces the presence of a rich development of elastic tissue in the mucosa and the muscularis of the stomach which he believes serves to maintain the tonicity of the organ. It is deposited generously at the fundus of the stomach, as he believes, to counteract the stretching which otherwise might occur at this part. He also finds it extensively deposited in the cardiac region where there is no proper sphincter muscle, and where it serves to keep the cardia closed through the increased tonicity thus provided the part. Mironescu notes the presence of elastic tissue in the muscularis mucosae and between the submucosa and muscularis. He also calls attention to the extensive distribution of elastic fibers in the region of the fundus, the cardia and also at the pylorus. lieves the tissue to possess great importance in sustaining gastric tonicity and mobility. Although it has not been shown that there is any special lack of elastic tissue in cases of gastric atony, it seems highly probable that such is the case, and that in gastrectasis depending upon atony we have to deal with conditions analogous to those existing in pulmonary emphysema. More recently through knowledge coming to us from radioscopy, we have learned that during functional activity the stomach is in a state of constant tension, through which the gastric contents is held under actual, though varying, pressure. It is easy to understand how the elastic tissue might play an important part in the performance. In order to comprehend the nature of gastric atony and certain cases of motor insufficiency, we must keep in mind this important function of the stomach, that is, the tonicity normally present in the stomach when at work. We must hold to the conception that when digestion is in progress, the gastric contents is under pressure produced by the walls of the stomach. It is in the absence of this pressure, due to the falling off of gastric tonicity, that we have the development of those phenomena constituting gastric

atony. But this loss of motor power depends not alone upon the deficiency of elastic tissue. It largely depends upon weak or degenerated muscles. The affection is therefore found to develop as a secondary manifestation of long-continued malnutrition, or more locally as the result of extension of inflammation from mucosa to muscle. We do not know to what extent the muscle coats are involved in cases of ordinary gastritis. We recognize that infection and inflammation extend to the muscle coats in certain cases, but it is safe to assume that the muscle weakness is not the result alone of myositis, but that it also depends upon laxity or decreased tonicity of the muscle. In those cases apparently when there is increased sensitiveness of the mucosa, the stomach is unwilling further to irritate it by the normal stress of full tonicity. Of course, this full tonicity is not invariably present, yet at times it is in excess, for irritation of the mucosa may induce a spasm of the stomach. This, however, is a condition that cannot be long sustained and as in the case of the colon and other parts, a period of spasticity is followed by one of relaxation and atony.

Hormones.—A new physiological conception of the motor activity of the gastro-intestinal tract has come about through the discovery of a hormone which provides a natural stimulus to peristalsis. Zuelzer states that he has succeeded in isolating this activating substance from the gastric mucous membrane at the time of the acme of digestion. Like other hormones it acts through the blood and, introduced into the circulation of animals, excites true peristaltic waves. This discovery is likely to modify considerably our conception of gastric and intestinal atony. It is possible that through this medium we may obtain light on the cases of atony which are associated with and, we believe, dependent upon,

neurasthenia, psychasthenia and nervous shock to which reference has been made in another chapter. It is a well-known fact that gastric atony is aggravated by general fatigue, nerve tire, worry and insomnia. It frequently accompanies headache and menstruation. We are not able to state how much of this depends upon the disturbances of innervation. Possibly the formation of the internal secretion may be unfavorably influenced through the nervous system.

Over-eating and drinking have been widely blamed for gastric atony, but probably upon insufficient ground. Over-eating undoubtedly contributes in producing symptoms when the stomach is already weakened. It is probably not a primary cause.

There is no special relationship between gastric secretion and gastric atony. Physiologically we know that an active secretion and a high acidity stimulate motion in the sense that the pylorus is made to contract forcibly. But on the other hand, in low acidity, as in achylia gastrica, the stomach is known to empty itself more quickly than usual. Therefore, although there is a close relationship between secretion and motion, there seems to be none between secretion and atony.

There is a contradiction between the conception that active gastric secretion stimulates the motor activity of the stomach and the actual clinical findings. Even in cases without apparent pyloric obstruction, in which there is found a plentiful supply of gastric juice, the emptying time of the stomach is often prolonged. This would appear to indicate that though the "milling power" of the stomach is probably greater with than without active secretion, the emptying function is not enhanced. A copious acid supply, as above stated, reduces mainly pyloric contraction. It is difficult to decide whether or not

active secretion occasions greater gastric peristalsis except by producing opposing pyloric spasm. With high acidity there is increased tonus and this antagonizes the intermittent relaxation which is necessary to the development of rhythmic peristalsis.

In the radiograms of the over-tonic stomach the peristaltic waves may be scarcely discernible. (See Radiograms VI and XII.) Finally gastric myasthenia may be present with either high or low acidity.

Relation Between Atony and Dilatation.—Considerable confusion has arisen as to the place which atony holds with relation to dilatation of the stomach. Apparently this has resulted from the unfortunate application of the term "motor insufficiency." This expression is used to denote a delay in the emptying of the stomach which depends upon myasthenia or weakness in the propelling part of the stomach, and it is also used to describe the tardy emptying of the stomach when this is the result of obstruction at the pylorus. In the latter event, the stasis may be the result not of insufficient motion but of excessive motion and contraction with consequent pyloro-In such an instance there is no true motor insufficiency. There is, of course, insufficiency in propulsion, but this arises not from a lack of motor power, but from pyloric spasm which the detrusor muscles of the stomach are not able to overcome. Whether it comes about from gastric myasthenia or from pyloric spasm, food stagnation is the natural result of retarded emptying of the stomach, and with food stagnation there is often associated dilatation. Hence when one speaks of dilatation of the stomach, it is necessary to inquire into the factors that lead up to this dilatation. Is it a dilatation which results from the progressive relaxation of an atonic stomach, or is it a dilatation which appears as the later

manifestation of pyloric stenosis which has led to a long-continued and progressive distension of the fundus of the stomach? Although food stagnation occurs in both instances, there is a wide difference in nature between the two conditions. Gastric atony, it is true, may supervene from prolonged over-distension, resulting from pyloric obstruction.

Temporary Gastric Atony.—It is well to emphasize the fact that gastric atony often occurs as a temporary matter, often as a sympathetic reaction to toxic, reflex and general nervous disturbances. This matter has been discussed elsewhere, but it must be borne in mind when dealing with the general subject of gastric myasthenia.

Symptomatology and Clinical Course.—As would be expected from the facts here set forth, the course of gastric atony is most variable, depending upon the etiologic sub-The patients are usually comfortable when the stomach is empty, and are uncomfortable soon after food is taken. A sense of heaviness in the epigastrium develops, there are gaseous eructations which are favored by a relaxed condition of the cardia, and fermentative conditions with infection of the gastric mucosa often oc-Therefore there is developed a set of symptoms based on auto-intoxication: a flabby, coated tongue; a disagreeable sense of taste; often coldness of the extremities with disturbed cutaneous secretions, and the patient complains of lassitude and mental confusion. Headache and the results of constipation and hepatic derangements may be added to the picture. Anorexia in the later hours of the day may be present, while in the morning, the desire for food may be unusually great. In other words, there is an irregular appetite. Perhaps too little is here said as to the local gastric symptoms. It is a question that must not be pursued too far because the local symptomatology is variable. In fact, the complaints sometimes are of general rather than of local symptoms. However the sense of weight already mentioned is a local symptom usually present. Even with moderate motor insufficiency and atony, although other symptoms attract little notice, the patient may suffer from disturbed sleep or insomnia. This is particularly true at middle age or later. Wakefulness and cardiac excitement lead the patient to seek relief and the less manifest disturbances of the stomach may be overlooked unless careful questioning is made to disclose the fact. There are present also slight pyrosis, eructation and a sense of gastric oppression.

When even these symptoms are absent it is sometimes found possible to overcome a troublesome insomnia by directing treatment to atony and motor weakness. Often the patient finds that by taking mint and soda, "ginger tea," or some other carminative, he is able to sleep whereas hypnotics are ineffectual.

With the oncoming of gastritis and fermentation, and with the presence of neurasthenia, there may be a large group of gastric symptoms, such as burning, acidity, pressure, pyrosis and even pain. All of these symptoms may be removed by lavage and the appetite may promptly return, but with the taking of food the symptoms recur.

Atony, transient and persistent, is often the cause of the complaints made by the dyspeptic and the convalescent, and a thorough understanding of the question is of practical importance.

Diagnosis.—Gastric atony is to be recognized by means of the stomach tube. Instead of emptying itself in four or five hours after a moderate repast, or seven hours after the taking of the Riegel test dinner, the stomach will be found to retain food much beyond this time. If

lavage is practiced during the morning fasting hours, considerable detritus from the meals of the preceding day may be found, although this is not common in uncomplicated atony. Such stagnation is often due to accompanying conditions, such as pyloric stenosis, or to a "water trap," or other deformity or displacement of the stomach. When morning stagnation is found, radiographic investigations should be made as well as those of the stomach tube. The remnant test of Mathieu and Remond for motor insufficiency, previously described, is especially applicable to the diagnosis of early cases.

Six or eight hours after the Ewald test breakfast 300 or 400 c.c. of gastric contents may be aspirated. It is important to note that whereas in lavage of the normal stomach, the gastric contents is returned under moderate pressure until the stomach is practically empty, this is not true in cases of gastric atony. Owing to the absence of tonicity of the stomach, the usual pressure upon its con-Therefore, the returning tents is no longer exercised. stream is started with difficulty. Siphonage is often interrupted and the stomach empties itself haltingly and imperfectly. In attempting to withdraw the contents for chemical examination after a test meal it is often impossible to procure undiluted contents without the assistance of the aspiration bulb. This contrasts strikingly with the result obtained upon using the stomach tube in that other type of motor insufficiency, so-called, that is, in cases of food stasis due to pyloric stenosis. With the latter condition the gastric tonicity is higher than normal, and the stomach contents is projected through the tube with such violence that one is at once assured that he is not dealing with gastric atony.

The fluoroscope betrays the presence of gastric atony by showing that the food is not held at first in the upper part of the stomach by its usual hopper-like action, but that it sinks to the lower part where it rests for a long time only slightly disturbed by the feeble peristaltic waves.

Prognosis.—The prognosis of gastric atony for complete recovery is poor in those cases which seemingly are due to the poor development of elastic tissue and muscle, that is, in the congenital cases. It is poor in those instances in which the atony results from long-continued gastritis. On the other hand the prognosis is good in cases which result from disturbed innervation, transient toxemia or transient infection. Atony following chronic infections is likely to persist and is an embarrassment in the treatment of many cases of tuberculosis. Atony following the acute infections requires long and painstaking treatment without which it may continue indefinitely.

Treatment.—In the treatment of those cases of gastric atony which are due to imperfect development and in which the condition may be considered as a somewhat necessary and natural state continued attention is required. All hygienic measures leading to a better physical development usually produce greater digestive efficiency. Graduated passive and active exercises with the assistance of massage are of very great importance. An outdoor life with walking, horseback riding and other recreations, so regulated that they do not produce overfatigue, is very beneficial. General innervation may be stimulated and greater general and gastric tonicity acquired by the judicious employment of the cold plunge, the needle bath, the cold spinal douche and the alternating hot and cold, or "tonic," douche over the body, especially over the epigastrium. Caution is necessary in the use of these measures. If applied carefully and

progressively, they are invaluable, but if thoughtlessly undertaken depression and greater atony will probably ensue. Considerable benefit attends the application of electricity. The static spark over the epigastrium and precordium, neck and spine should be tried, and intragastric faradization daily for a considerable period materially increases the tonicity of the stomach as I have shown in many cases. The alternating hot and cold intragastric spray has been recommended, but the result obtained is hardly worth the trouble. For the atony of convalescence there must be some modification of these measures. Care must be taken not to overdo local and general stimulation. Patience should be exercised and nature allowed time for recuperation. Meanwhile, by the use of a suitable dietary and appropriate medication, we may prevent an increase in the atony.

DIET.—The diet is of importance. If too large an amount is given at one time the stomach sometimes appears to become discouraged and symptoms at once develop. If we feed too frequently in small quantities, the stomach does not have time to empty itself and may be-Therefore the quantity and the interval come resentful. must be suited to each case after actual trial. No hard and fast rules can be made. Individual cases differ also as to the kind of food that agrees best. The best results are sometimes, but not always, obtained with a liquid diet. Foods must be carefully prepared, a statement which is a solecism and yet is of great importance. Patients will sometimes take with no resulting disturbance small quantities of fresh vegetables carefully prepared. Yet they will suffer at once when they take vegetables which have been too long in the market, improperly prepared. Such patients are almost always injured by fresh bread, although bread that is heated over is more likely to agree than cold bread. If there is a fair secretion of hydrochloric acid, tender meats may be allowed, but in that case the meal should be practically confined to meat, to the exclusion of milk and carbohydrates. Carbohydrates and peptonized milk may be selected for one or two meals of the day and the digestion of the former may be facilitated by the use of extract of malt. The cooked fats are usually an abomination in these cases, vet oil or butter used with the food is sometimes well borne. Often it is desirable to give the foods very hot and they are better borne if there is permitted a moderate amount of a condiment such as horseradish or some stimulating beverage, such as ginger ale, Apollinaris water or, in exceptional cases, sparkling wine. A teaspoonful of cognac or creme de menthe may be taken at the end of the meal, and some cases are benefited by a demi-tasse of black coffee.

MEDICAMENTS.—In cases of simple and especially transient gastric atony prompt benefit is found from taking the carminatives such as cardamom, annis, peppermint, wintergreen or ginger. Many preparations that have achieved a reputation in dyspepsia are effective in proportion to their power to whip up gastric motility; hence the benefit that sometimes attends the addition of capsicum to the food. In the use of these remedies, as in the use of alcohol or of the spirits of ammonia, we must be careful not to excite a gastritis. It is much more difficult to treat atony and gastritis associated than it is to treat either alone. Nux vomica may be given temporarily in large doses. I like to give the powdered or rasped nux vomica in doses of one or two grains (.06-.12) gradually increased to five grains (.30) to be taken half an hour before meals. This dose is too large for some individuals. Its administration should be continued for

two or three weeks, and then succeeded by an interval of rest. It is highly probable that the hormone which stimulates gastro-intestinal peristalsis will prove to have real value in cases of gastric atony.

# CHAPTER XX

### GASTRECTASIS. DILATATION OF THE STOMACH

Twenty years ago much more was said about dilatation of the stomach than is to-day. As knowledge of the pathology of dilatation has advanced consideration of the subject has become simplified. Bouchard attributed to gastrectasis the source of most so-called functional disturbances. He believed that moderate stasis with resulting fermentation of gastric contents produced intoxications having a wide array of manifestations. Though we recognize the great importance of gastric dilatation, Bouchard's view of the subject is not at present admitted. Concerning the use of the term "dilatation of the stomach," there is, strange to say, some disagreement.

We recognize that dilatation is a natural result of gastric atony and also of long standing pyloric stenosis in which the stomach has begun to yield after continued and ineffectual effort towards evacuation. It came to be seen that it is not so much the dilatation of the stomach that causes trouble as it is the food stagnation and the motor insufficiency which accompany the gastrectasis. Thus there grew up a conception of dilatation of the stomach which became confused with that of motor insufficiency. Riegel and his followers paradoxically spoke of a gastric dilatation in which the stomach was not dilated, but in which there occurred, owing to motor insufficiency, the symptoms which belong to dilatation. In defense of this position, Riegel claimed that it was not a question of the size of the stomach (gastromegaly), not even a question

as to the degree of dilatation. The matter of real importance, he maintained, was the presence of a group of symptoms which depends upon the motor insufficiency. He found that these symptoms might be present when the stomach showed no increase in size; in fact, sometimes, owing to increased tonicity, the actual containing power was less than that of a normal stomach. Yet these cases presented the symptoms of dilatation, and for that reason he spoke of them as such. The question is an illustration of the importance of a correct use of terms.

Gastromegaly.—We sometimes find an extremely large stomach with motor function unimpaired and consequently no symptoms. Gastromegaly may be a congenital peculiarity, or it may result from the eating of coarse foods in excess or from the drinking of large quantities of beer. Such cases can scarcely be considered to be dilatation of the stomach.

Dilatation from Atony.—This is fully described in the chapter on Atony of the Stomach.

Dilatation from Stenosis.—As a result of long-continued pyloric obstruction with unnatural and relatively ineffectual motor efforts, dilatation of the stomach is pro-The dilatation is preceded by hypertrophy of the There is marked increase in the intragastric muscle wall. pressure. After a time the less resisting regions of the stomach wall give way and the stomach dilates in a manner closely resembling the dilatation of the heart in aortic stenosis, or marked arteriosclerosis, with high blood pressure. We formerly spoke of this heart condition as one of simple hypertrophy finally developing into hypertrophy with dilatation and possibly ending in dilatation in which little evidence of hypertrophy remained. The same facts may be instanced in the urinary bladder with prostatic obstruction. Now all this seems clear enough, but what are

we to say of the cases in which there is muscular hypertrophy and yet in which the cavity of the stomach has not increased? To my mind this cannot be classified as dila-The stomach is either dilated or it is not. When it is not dilated and yet when there are present the symptoms often seen in cases of developing dilatation, we must attribute these symptoms to their real source; that is, to pyloric obstruction and to the efforts on the part of the stomach to overcome that obstruction. Motor insufficiency is a poor term with which to describe this condition for its meaning is confused with that of a very different condition, namely, myasthenia gastrica. ever, the term has been so long employed that it is in no man's power to displace it, but I hope in this description to assist in the understanding of its meaning.

In addition to the two types above-mentioned of gastrectasis, that from atony and that from obstruction, differing widely in nature, there is a third form, that of acute dilatation of the stomach. Because it can best be described by itself, it will receive later consideration.

Atonic dilatation although it is closely related to atony, is sometimes more than atony. The latter condition may continue for a long time without the development of dilatation; the stomach may be said to be in a state of relaxation. Dilatation refers to a condition in which the stomach is habitually expanded and in which its capacity is greater than can be accounted for merely by relaxation. A relaxed stomach may almost instantly, through the resumption of its normal tonicity and contractility, resume its usual shape and size, and at times the relaxation may be considered rather as a physiologic condition. Dilatation refers to a pathologic enlargement associated with disturbance of motor function.

Etiology.—The causes of atonic dilatation, in addition

to those which relate to relaxation may have to do with degeneration of the muscular tunic of the stomach, a condition which may result from gastritis or any other factor leading to muscle degeneration. Apparently dilatation at times depends in some obscure way upon disturbed innervation. We find that dilatation develops in sufferers from psychic disease or shock. There are well authenticated instances in which it has followed a single blow upon the epigastrium; besides this, we may obtain a hint as to nervous causes of atonic dilatation in the study of those cases of acute dilatation which result from surgical shock or very acute illnesses not directly connected with the stomach. This is not to deny that the causes that lead to atony may be sufficient if long enough sustained to give rise to dilatation. We are not able to distinguish between the etiologic factors at work. only differences in these are probably those of degree and of duration rather than those of kind. Atonic dilatation occurs much less frequently than that form which follows obstruction.

The causes of dilatation from obstruction may be easily understood. If the stomach is well nourished, if there is no defect in blood supply and innervation and no dyscrasia interfering with the nutrition of the gastric parietes, there would naturally result, when obstruction occurs at the pylorus, an unusual effort, therefore an overdevelopment on the part of the muscle of the stomach. This gastric hypertrophy may for a time be sufficient to overcome the pyloric narrowing. Under these conditions, the stomach may be able to empty itself without great delay, but the patient suffers as the result of the unusual gastric effort. The situation suggests that of a small ship with a large engine. In the course of time more energy is demanded than can be supplied from

the nutrition available through the gastric blood supply. The organ becomes fatigued; its expulsive power decreased and stretching at the weakest point is the natural result. In old cases we may find that certain regions of the gastric wall have yielded more than others.

Anatomical Changes.—Sometimes in dilatation with stenosis we find greatest dilatation at the antrum pylori; more frequently this occurs in the fundus and along the greater curvature. An explanation for this may be found from the study of the normal variations in shape and motility of the stomach occurring under physiologic conditions. We learn from the admirable studies of Cannon that during digestion the upper part of the stomach assumes a pouch-like expansion in which it acts to the lower part of the organ something like a "hopper." lower third of the stomach assumes the shape of a tube with a remitting and recurring constriction where the middle meets the final third of the stomach, by means of which portions of the pabulum are held under a certain degree of pressure, thus favoring movement towards the pyloric extremity. The physiological stress both of motion and of secretion appears to be greater in the last third of the stomach. This has a bearing on the more frequent appearance of inflammation, ulceration and malignant disease in this portion of the organ and it doubtless accounts for the dilatation which, notwithstanding the thickness of the muscle coat, is sometimes observed in this region. The walls of the stomach as a whole are not necessarily thin in this type of gastrectasis, although where the dilatation is most pronounced the walls show greater thinning than at other parts. Atony may develop and then degeneration occurs in the muscle coat, and the walls of the stomach are correspondingly thinned.

Clinical Course and Symptoms.—In a slowly progress-

ing dilatation the condition gives rise to symptoms that have been described already in the discussion on Reichmann's disease, the symptoms of beginning motor insufficiency. In atonic dilatation this state of affairs may continue for a long time before the more marked evidences of food stagnation develop. With the further disappearance of contractility and resulting weakness of the walls of the stomach, the food accumulates in considerable quantity, the sensation of weight and fullness is redoubled, loss of appetite and nausea develop. These symptoms are relieved when the stomach is emptied, as is shown in the effect of vomiting or of lavage.

In dilatation secondary to obstruction, in which type of the disease gastrectasis is seen in its fullest development, the most marked symptoms are observed. The patient at first suffers as in Reichmann's disease but soon from the accumulating contents the dilated stomach becomes distended, and its efforts to empty itself give rise to distressing symptoms. This is often accomplished by thirst and after a while vomiting is produced, when an astonishing quantity of fermenting and foul-smelling material may be ejected. The quantity of vomitus is somewhat dependent upon the degree of dilatation. This vomiting may occur only once in two or three days and then there appear remnants of the coarser foods that have been accumulating during that time; seeds, the fibrous portions of fruits and vegetables, the more tendinous portions of meat are discharged and one may recognize substances which had been swallowed days, weeks and occasionally months before. This depends upon the fact not merely that vomiting has not occurred, but that the stomach has not been fully emptied for a long time. These bizarre results are generally observed in old cases in which lavage has not been previously practiced. The odor of the stomach contents is characteristic and serves during lavage to identify a case of food stagnation even at a considerable distance from the patient. There is a musty, "mousey" quality to the odor that is not found in other conditions. Lavage affords the greatest comfort and appetite quickly returns. Thirst results from the accompanying over-secretion and the withdrawal of fluid from the organism. Absorption of water from the stomach during food stagnation is slight, while the discharge into the stomach of the water of gastric juice is considerable, so that in course of time the fluids of the body are greatly diminished as is shown in the concentrated urine, the dry mouth and tongue, the offensive breath and the sodden skin. Acidosis develops and the patient rapidly undergoes emaciation. In advanced cases the loss of flesh is more extreme than I have seen in other ambulatory patients. Owing to the long-continued denutrition accidental infections occur which may mislead the patient as to the nature of his trouble. one case of long standing gastrectasis the patient also suffered from bronchitis, and came seeking advice as to the most suitable climate for the relief of a supposed pulmonary phthisis. His lungs were sound and his cough disappeared under treatment directed towards the correction of gastric dilatation.

The characteristic of the vomiting is that it usually occurs at comparatively long intervals; the frequency depending upon the amount of the ingesta and of the oversecretion. At first there is vomiting once or twice a week, and as the trouble increases, perhaps once in twenty-four hours. The second characteristic of the vomiting is the large quantity that is ejected and the third, the peculiar odor. When gastritis co-exists with gastrectasis, the stomach may become so irritable that it declines to hold

any considerable quantity, and the vomiting is more frequent. With this frequency there is, of course, a falling off in quantity, and the peculiar odor is less noticeable. But even when the emesis is frequent and the vomitus small in amount, something of the characteristic odor still persists. In children and in adults with dull sensibility, the desire for food seems to be but little depressed. Before vomiting becomes conspicuous, eructations of foul-smelling gas and regurgitations of acid stomach contents may be expected. These are among the earliest symptoms and are generally accompanied by a sensation of gastric unrest and sometimes by spasmodic pain. Almost always there is constipation with small, dry stools.

Diagnosis and Objective Symptoms.—Conspicuous among the objective symptoms is emaciation. At the same time, one may perceive that the upper or left upper part of the abdomen is over-prominent. Though it is observable when the patient is in the upright position, placed on a table, lying upon his back with the abdomen uncovered, even greater fullness is evident in the upper abdomen. The location and extent of this field of prominence depends upon the size of the stomach and is greatly modified with the oncoming of ptosis. In the presence of the last-named condition, the dilated stomach may show itself by a prominence in the lower or lower left abdomen rather than in the upper quadrants. In dilatation depending upon atony this area of fullness rests without motion or sound, and there is an absence of the sounds of gastric peristalsis even with the careful practice of auscultation. The findings are very different when the dilatation is the result of stenosis and is accompanied by hypertrophy of the muscle coat. In the latter type of gastrectasis one may easily hear splashing which is produced by the involuntary and rhythmic movement of the stomach as the contents is hurried from the upper two-thirds into the lower third of the stomach and thence propelled back. What is taking place under cover of the walls of the abdomen becomes very evident to the onlooker even without use of the fluoroscope. The con-



Fig. 42.—Gastrectasis with Secondary Emaciation.
(From Riegel. Diagnosis and Treatment of Gastric Dilatation. Modern Clinical Medicine.)

dition affords an excellent opportunity for the visual study of gastric peristalsis. The walls of the abdomen conform closely with the movements of the walls of the stomach; a fullness bulges from beneath the left costal border and rolls like a ball across the abdomen, towards

the pylorus and there, under increasing tension, it remains for a moment, then the fullness subsides with the upward projection of the contents. To the unprofessional eye, these movements suggest the effort of some living thing attempting to escape. These vigorous manifestations are accompanied by unpleasant subjective symptoms of tension and torsion. When the stomach is only moderately distended or when it becomes fatigued through prolonged efforts, or when in long standing cases, denutrition and exhaustion have overcome the muscle power of the stomach, these visible movements are less conspicuous, but are usually observable. This manifestation, first described by Cruveilhier, is a most important lesson in the morbid physiology of the stomach and it is surprising that its nature and even its existence remained long unknown in general medicine. When these movements are not perceptible they may be stimulated into showing themselves through the sudden application of the cold hand over the epigastrium, by dashing on a little cold water or by gently tapping over the region of the stomach. If there is needed any proof of the relief to spasmodic condition of the stomach through the local application of heat, it may be demonstrated in this condition, for hot fomentations almost uniformly subdue these visible movements and reduce the stomach to a state of motor tranquillity.

Palpation.—Palpation reveals the presence of distension or of over-rigidity of the stomach. It is not necessary to repeat what already has been said on this matter in the section on Pyloric Stenosis. Much may be learned concerning tonicity of the walls of the stomach, and the extent and consistency of its contents, by conjoined palpation and percussion. With the fingers slightly curved, the tips are suddenly brought down upon the

surface of the abdomen with force just sufficient to displace at that point the fluid contents of the stomach. This act must not be performed too quickly, for though the pressure of the fingers is to be made suddenly, it is also to be made progressively and increasingly, thus allowing time for the contents to flow away beneath the pressure. It is well in practicing this method to begin at a point known to be below the lower border of the stomach and to continue making these little dabbing motions, striking the abdomen each time at a point somewhat higher up, so as to recognize the difference in the sensation conveyed to the palpating hand. This method which has been emphasized by Cohnheim is of great importance in the physical examination of the stomach.

THE STOMACH TUBE.—The stomach tube is of importance in diagnosis of gastrectasis. With practice one learns to judge the lower border of the stomach by noting the manner of reflow and the character of the contents removed. By measuring the length of the tube introduced beyond the incisor teeth necessary to reach the lower border of the stomach, it is possible to estimate the degree of dilatation or ptosis that is present; also, by the character of the contents, we can judge the degree of To make this latter of more definite food stagnation. value we must have recourse to test meals. The stomach having been emptied on the preceding night, the Ewald test breakfast should be given and the quantity returned one hour later ascertained. While from a normal stomach we aspirate from 30 to 100 c.c. of thin contents, in case of dilatation from 500 c.c. to 1000 c.c. may be withdrawn. This large quantity depends partly upon the lack of emptying power in the stomach and perhaps also upon over-secretion. When over-secretion is a prominent feature there may be withdrawn from the stomach double the

quantity taken for the test meal. A normal stomach should empty itself of an Ewald test meal within three hours of its ingestion, and of an ordinary meal within five or six hours. Riegel was in the habit of examining the stomach seven hours after his test dinner, at which time the stomach should be empty. In dilatation with impaired motor power, the stomach is scarcely ever empty, but the degree of stagnation may be estimated by postponing further and further each day the hour for aspirating the stomach after a test meal, thereby measuring in time the degree of motor insufficiency. In cases of moderate insufficiency, it will be found that the stomach is nearly empty in the morning, containing merely the detritus of the evening meal. Of course, in a normal stomach even this should have passed on. This sign is of importance in the study of cases of motor insufficiency, and to make the matter more definite, it is advisable to give with the evening meal a spoonful of dried currants, a small quantity of flaxseed or some similar insoluble food. Should there be a delay in the emptying of the stomach, these substances will be found remaining after the more soluble ones have passed the pylorus. When there is high gastric acidity the digestion of starch is delayed beyond that of proteids. When the solution of the gastric contents is complete, the proportion of retained carbohydrates may be determined by using the fermentation tube as suggested by Strauss. Occasionally the obstruction which gives rise to the dilatation instead of being located at the pylorus is really present in the duodenum. Location below the bile papilla may be recognized by the presence of bile in the stomach contents or in the vomitus. The use of salol, iodopin or oil for the study of the motor efficiency is scarcely necessary. Much time is spent with these tests which might

better be employed in learning the full resources of the stomach tube.

It is in case of dilatation that there sometimes develops the interesting condition known as gastric tetany, a subject that will receive attention elsewhere. Whether its occurrence depends on the over-withdrawal of fluids from the body, upon reflex action, upon disturbance of parathyroid secretion and the calcium balance or upon auto-intoxication, is a question. The first manifestations of tetany often occur soon after the practice of lavage. the reason for which is not clearly understood. know that auto-intoxication occurs in dilatation of the stomach, although less frequently than was believed by Bouchard. Probably this in part is the result of star-. vation and the development of acidosis and of other metabolic faults. When food stagnation is pronounced, the urine becomes very scanty but it increases in quantity directly the motor power returns.

It is to be understood that there are no variations in gastric chemistry that are characteristic in gastrectasis.

The one characteristic in the stomach contents to be revealed by lavage is the evidence of motor insufficiency; in other terms, some degree of food stagnation.

DIFFERENTIAL DIAGNOSIS.—Gastrectasis is to be distinguished from gastroptosis by the presence of ischochymia; but it is to be remembered that both conditions often coexist. It is by the results of radiography and fluoroscopy that the joint occurrence of dilatation and downward displacement can be best understood.

Transient ischochymia, resulting from either temporary spasm or brief motor inaction, is to be recognized by repeating the examination.

Prognosis.—The fate of gastrectasis depends upon its cause. A proportion of cases which result from pyloric

obstruction can be greatly relieved and sometimes cured by medical treatment. When the pylorus is encircled with contracting fibrous tissue or when a growing tumor increasingly encroaches upon the passage, or when the cicatrix of deep ulceration progressively narrows the canal, relief must wait upon surgical intervention. The treatment of the dilatation becomes practically that of stenosis, and need not be repeated. The outcome of those cases which depend on atony is uncertain in ratio with our inability to remove the underlying causes and these may be permanent or transient.

Treatment.—Except in operative cases, there is no satisfactory treatment of gastrectasis without the use of the stomach tube, but it is surprising to note the improvement that almost immediately follows upon adoption of the plan of washing the stomach clean at night and allowing it to rest until morning. Lavage too often repeated is objectionable, for thereby the patient may be depleted. In advanced cases there may be withdrawn from the stomach more than is ingested. There is something besides aliment lost through oft repeated gastric washing; there is harmful abstraction of water, which in case of over-secretion is a matter of consequence; the organism is deprived of ferments and other substances that should be economized. Therefore lavage should not be practiced indiscriminately, although a lack of intelligent frequency is also to be condemned. I have observed patients with a static narrowing of the pylorus who have found it advisable to practice lavage at intervals during a period of years, from which it may be inferred that there is little danger in judicious use of the method. The fact is that it is less harmful in some cases than in others, and we do not fully understand the reason for this. Next in importance to lavage

and necessarily to be considered with it, is the question of diet. The character of the food should be such that it provokes the least spasm and retention in obstructive cases and such as does not require too much motor activity of the atonic stomach. Application should be made of the same principles of treatment described in the article on gastric atony, associated when necessary with lavage.

# CHAPTER XXI

#### ACUTE GASTRECTASIS

During the last few years among both American and foreign observers, increased interest has been aroused in the subject of acute gastrectasis. The condition follows accidental or surgical injury and also in some instances grave constitutional conditions.

Brown and Seidel,1 after experimental research, conclude that acute dilatation of the stomach depends upon some lesion of the nervous apparatus, that it is the result of acute gastric insufficiency arising from a functional, rather than from a mechanical difficulty.

Lewis A. Conner<sup>2</sup> has contributed a valuable study of the disease, adding a new group of cases, in which he would show that dilatation results from sudden obstruction produced by torsion of the mesentery resulting from displacement of the viscera, the displacement causing compression of the duodenojejunal juncture of the gut. Conner emphasizes the importance of spinal deformity as leading to visceral displacement and hence to obstruction. Recently Roussel<sup>3</sup> has reported a case following typhoid fever, the patient having also been prematurely delivered of a child, the gastrectasis appearing late in convalescence.

Kemp 4 of New York recites additional cases and reports experimental work on animals. His view corre-

<sup>&</sup>lt;sup>1</sup> Arch. f. Verdauugskr., Aug., 1907. <sup>2</sup> Am. Jour. Med. Sci., March, 1907. <sup>3</sup> Med. Rec., Jan. 25, 1908.

<sup>4</sup> Am. Jour. of Surg., Nov., 1908.

sponds to that of Conner in attributing the disease to a sudden obstruction, but there is apparently some confusion as to whether the obstruction causes the distension of the stomach or whether the distension through displacement leads to the obstruction. Both of these observers attribute the dilatation to distension. Indeed the cases reported by Kemp seem to be instances of aerophagy. Dr. Paul Tissier of France relates three personal experiences from which he deduces that acute distension,

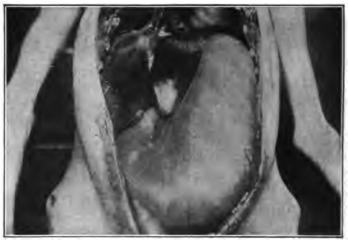


Fig. 43.—Acute Dilatation of the Stomach from Obstruction of the Duodenum. The patient had diffuse tuberculosis of the liver and spleen.

(New York Hospital Collection. Permission of Dr. Conner.)

involving the stomach and sometimes the intestine, represents the pathology of the condition. In discussing this Linossier makes the assertion that the sudden and acute distension of the stomach was scarcely explainable without admitting a state of paralysis to have preceded it, a conclusion which Tissier was willing to accept. It is extraordinary that greater upward escape of gas does not occur especially in view of the fact that some cases are accompanied by vomiting. Of course the vomiting

may be produced through spasmodic contraction of the diaphragm and abdominal muscles, even with the stomach in a state of complete paresis, but one would expect that when liquid gastric contents is discharged, that gas would also be ejected.

Boas 5 divides the cases into the following classes: (1) paresis after errors in diet; (2) after severe infectious diseases; (3) after exhausting chronic diseases; (4) after traumatism; (5) after laparotomies; (6) from chloroform narcosis alone (central paralysis); (7) from the mechanical kinking of the pylorus or the jejunum, or (8) from acute obstruction.

In addition the disease has been ascribed to pyloric spasm from dietary indiscretion and to deformity of the spine. Finally, there are cases the development of which has so far remained unaccounted for.

When too many causes are evoked in explanation of a definite and frequently observed departure from a normal physiologic state, it may be assumed with safety that the rationale is not known. What actually occurs in these cases is a profoundly over-distended stomach, stretched by the accumulation of gas and fluid which is in part The condition appears to be identical with the rapidly fatal wind colic observed in horses. It seems difficult to explain in obstruction coming on simultaneously in the duodenum and at the cardia without preceding paralysis of the stomach. Admitting such paralysis, one may conceive that a spasm would be excited in the tubes leading to and from the stomach. Granting the obstruction from spasm, we are able to explain the oncoming distension and rapid dilatation, and there is ground for believing that such a state of the stomach would produce still further mechanical obstruc-

<sup>&</sup>lt;sup>5</sup> Boas, "Disease of the Stomach," American edition, 1907.

tion at the duodenojejunal juncture. This would seem to be the most rational explanation of this remarkable symptom-complex. Although the condition as recognized includes the element of distension with gas, this is occasionally absent in instances of acute atony with dilatation; one or more liters of liquid are found in the stomach, yet very little gas and no actual distension. These cases accompany shock and are apparently related to acute dilatation. Although vomiting occurs, the condition is not to be demonstrated without the use of the stomach tube.

My first case of acute gastric dilatation was seen twenty-five years ago in a child eleven years old approaching death from acute tubercular pneumonia. Aside from the abdominal distension, which occurred shortly before death, there was nothing to attract attention to the stomach. At autopsy I was surprised to find the stomach distended with gas and dark-colored fluid, and capable of holding more than four liters. No mechanical obstruction was noted. This case shows that acute gastrectasis may develop in childhood, whereas, most cases reported have occurred in middle life. Fussel 6 draws attention to the frequency with which the condition occurs in pneumonia.

Another case was that of a man of sixty who was entertaining some friends in the evening at a little supper. He was attacked with terrific pain in the abdomen, thoracic oppression and shock. He became pulseless and quickly succumbed apparently from heart failure. The stomach was enormously distended without apparent cause.

A number of cases seemingly die from the effects of pressure upon the heart; in these tachycardia is a nota-

<sup>6</sup> Am. Jour. Med. Sci., Dec., 1911.

ble symptom. The appearance of the disease after laparotomy led to the belief that it was the result of abdominal shock, but as cases accumulated it was seen that severe injury to any part of the body, not alone the abdomen, might induce the condition and as shown it may follow any grave constitutional disease. As a rule the symptoms develop rapidly, in operative cases usually within twenty-four hours. However, in some cases following operation, the dilatation does not occur until several days have elapsed. There is usually abdominal distension sometimes extending from the ensiform to the pelvis, persistent vomiting, profound depression or collapse, shallow breathing, very weak and frequent pulse, subnormal temperature, and scanty urinary secretion or anuria. In 50 per cent of the cases pain or tenderness is present and in the majority there is constipation, although in a proportion diarrhea exists. In some cases vomiting does not occur, and then the condition is the more readily overlooked. There is generally dullness upon percussion over the abdomen, but at times there may be tympany. The French have shown the importance of radiography in the diagnosis of the condition. Of 102 cases collected by Conner, 74 died, giving a mortality of 72 per cent. The diagnosis ought not to be difficult provided one is acquainted with the disease. It may be verified readily by the use of the stomach tube.

Treatment.—As a rule great improvement immediately follows the practice of lavage which sometimes has to be repeated frequently owing to the rapid re-accumulation of fluid in the stomach. Alimentation should be practiced only per rectum. Attention to posture has proved valuable. The foot of the bed should be raised, or in some way the patient should have the lower end of the trunk tilted upwards so that drainage will be towards the esoph-

agus, and so that tension may be removed from the duodenojejunal juncture. The dorsal decubitus seems to favor the continuance of the trouble, and for this reason Snitzler conceived the idea of relieving the symptoms by causing the patient to lie on the belly, an expedient that proved to be successful. Similar results have followed this treatment at the hands of others, and it is apparently useful method. Hypodermics of strychnin physostigmin have appeared to be of advantage and are indicated; also hypodermoclysis, enteroclysis, and stimulation per rectum. In cases properly treated the results have been very satisfactory. Unfortunately the condition is too often overlooked. Undoubtedly grave cases prove fatal even without the development of extensive dilatation. Following trauma, surgical or otherwise, in pneumonia or other grave disease when the patient shows evidence of shock and collapse that cannot otherwise be accounted for, the possibility of beginning acute dilatation of the stomach should always receive careful consideration.

Repeatedly I have been asked to explain the presence of persistent vomiting, circulatory depression and prostration occurring in patients immediately after anesthesia, when convalescing from an operation or during some serious illness, and have found the symptoms to depend upon acute gastric dilatation.

Usually there could be detected in these patients on palpation an unnatural resistance in the upper zone of the abdomen especially on the left side; generally there was obscuration of the cardiac apical impulse. The presence of the distension is often overlooked because of concealment by surgical dressings, or misinterpreted for distension of the intestines.

When gastric distension exists or even when its exist-

ence is suspected, the stomach should be washed out repeatedly. This is emphasized because there is often a fatal delay in performing the lavage or it is not repeated often enough. The latter error depends upon the fact that less fluid is aspirated and less gas escapes through the tube than is commonly supposed to be the case in this condition.

# CHAPTER XXII

### AEROPHAGY AND GASEOUS DISTENSION

Some observers state that aerophagy or the swallowing of air is a factor of importance in the causation of acute gastrectasis. Granting that aerophagy exists, it would act as a contributing factor, but more than this can hardly be admitted. The swallowing of air and the consequent gaseous distension of the stomach occur in two distinct classes of patients. In one it depends upon the excessive secretion and frequent swallowing of saliva, which carries with it into the stomach a considerable quantity of air. In this class of patients the actual trouble is sialorrhea and the accumulation of gas is a mere The other class of patients are hysterical and incident. swallow air in quantities and reject it with noisy belch-The condition has been referred to in the chapter on Gastric Neuroses. A counterpart of aerophagy exists in certain horses that are called "cribbers." The chief symptom of the condition is a consciousness of gastric distension which is relieved by eructation. On physical examination there is a visible fullness under the costal arch with tympany upon percussion. When the stomach is in a state of tension, metallic tinkling sounds, especially marked when the patient swallows fluid, may be heard upon auscultation in the left subcapular region, in the infra-axillary region and over the stomach in front. In exceptional cases when eructations do not occur and in which the stomach becomes over-distended, relief is easily procured by the introduction of the stomach tube

through which the gas is rapidly discharged. The free end of the tube should be kept in a vessel of water so that an approximate idea may be obtained of the amount of the escaping gas and, by using a suitable bottle, the gas may be collected for examination. There is a common belief that this gas originates in fermentation, which is sometimes true but more often a mistake. It should be borne in mind that a certain amount of gas is natural to the stomach and without its presence the movement of the gastric contents would be somewhat embarrassed. The eructations of gas, as hitherto explained, depend upon a state of over-tonicity and motor excitability of the organ. Gas may originate from fermentation, in which event there are present in the stomach gas-forming bacteria in large number. It is a mistake to suppose that such microörganisms do not flourish when there is an active secretion of hydrochloric acid. It is a somewhat extraordinary fact that we find more bacteria. when secretion is present than when there is anacidity.

In achylia gastrica the stomach contents is often remarkably free from bacteria. One reason for this lies in the fact that when there is hyperacidity there is usually a certain amount of pyloric spasm which results in retarding the passage of food, that is, in partial stasis. It is a rule that stasis anywhere in the alimentary tract predisposes to fermentation and to gas formation. It is probably for this reason that gluttons and hasty eaters suffer more than others from the presence of gas, since an undigested meal is usually slow in leaving the stomach. Acetic acid fermentation accompanied by gas generally occurs in presence of hydrochloric acid, whereas lactic acid and butyric acid are formed in hypochlorhydria, and hence in cases of cancer. At times the gas has a

524

butyric odor or that of sulphureted hydrogen. Occasionally there is an excess of hydrogen or marsh gas, which may be ignited as it is discharged through the tube. cases of aerophagy, the elements composing the gas are in somewhat different proportion than in atmospheric air. There is less nitrogen and an increase in carbon and oxygen. Hydrogen and carbonic acid gas may result from fermentation. The diminution in oxygen is perhaps the result of absorption. There are some curious cases in which patients suffer from somewhat sudden over-distension of the digestive tract with gas, and after a short time this disappears without noticeable ejection. Dr. Woods Hutchinson has suggested that the gas may reach the alimentary canal from the blood and again be absorbed, somewhat after the manner of the filling and emptying of the swim-bladder in the fish.

Treatment.—It will be seen that the rational treatment must vary according to the origin of the gas, and attention may be necessary for the cure of sialorrhea and of hysteria if we expect to overcome certain forms of aerophagy; also a different diet and different remedies are required for overcoming the gases of fermentative origin. Where acetic acid is in excess the patient may be cured by conforming to a strict albuminoid diet and in butyric acid fermentation by eliminating fats, especially cooked fats, from the diet. The most practicable remedy is salicylic acid or the salicylates in repeated small doses, for instance magnesium salicylate .30 gm. (gr. 5). Other useful agents are bismuth salicylate, salol, hydronaphthol and benzoic acid, and for some reason not perfectly understood charcoal affords considerable relief.

The carminatives, stimulating gastric motility, are especially indicated when the symptoms depend upon spasticity of the cardia.

When there is marked distension of the stomach with gas, the stomach tube should be passed, thus allowing the evacuation of the stomach as in cases of acute gastrectasis.

# CHAPTER XXIII

### GASTRIC TETANY

This term is applied to tetany occurring in certain cases of gastrectasis. Although our knowledge of gastric tetany dates back to Kussmaul's original description of the use of the stomach tube in the treatment of the gastrectasis, the precise nature of the phenomenon is not yet understood. Kussmaul suggested that tetany might result from the dehydration of the blood, which is very conspicuous in cases of advanced dilatation, and likens the attack to the spasm seen in cholera patients. Others have thought to explain tetany as a reflex phenomenon and still others have attributed it to anto-intoxication. More modern researches have shown that the various forms of tetany are in some way dependent upon an insufficiency in the secretion of the parathyroid glands. It is known that the parathyroid bodies exert a regulating influence upon the calcium metabolism of the body. MacCallum showed that the introduction of calcium salts exercised a controlling effect upon the spasm of experimental tetany. This led to a hope that we had found a means of successfully treating this rare complication of dilatation of the stomach. However, experience shows that though calcium will promptly relieve the symptoms, especially when introduced intravenously, its action is not fundamentally curative. Kinnicutt in 1909 reported before the Association of American Physicians a typical case of tetany in which he employed preparations of parathyroid bodies, intravenous normal saline

solution and calcium salts. He concluded that no effect was observable from the parathyroid preparation, and but little from the normal saline injections but that the calcium salts exercised a promptly controlling effect upon the tetanic symptoms.

Two guinea-pigs injected subcutaneously with filtered stomach contents obtained through a stomach tube developed spastic conditions with clonic contraction and other nervous disturbances. The parathyroid bodies, so far as could be determined by histological examinations remained perfectly healthy.

Kinnicutt's description of the clinical picture of the tetany is as follows: "Both legs are held straight and stiff, the feet in extreme extension, the toes in plantar flexion. The muscles of the calves of the legs are hard. tense and drawn, the spasm is continuous, but pressure on the nerve trunk and blood vessels increases, if possible, the severity of the spasm (Trousseau's sign), and the same phenomenon is obtained on tapping over the motor nerve supplying the affected muscles (Chvostek's sign). The forearms are flexed at right angles on the arm, the wrists flexed to an extreme degree and cannot be extended, the fingers flexed at right angle at the metacarpophalangeal articulation and held rigidly straight in adduction, the thumb tightly against index finger not drawn into the palm. The spasms are continuous but, as in the case of the lower extremity, they can be increased both by Trousseau's and Chvostek's methods. The tendon reflexes appear to be about normal in both upper and lower extremities. There are continuous twitchings of the muscles of the face, which are increased by tapping over the course of the motor trunks. A slight degree of trismus is present. The musculature of the trunk is free from spasm which cannot be excited by mechanical irritation of the motor nerves or the muscles. The tachypnea is striking, the respirations being 80."

My own two cases were of the comparatively mild type and both recovered. In each there was marked gastrectasis and in one, a child five years old, gastric movements were noticeable through the abdominal wall. The other case was that of a woman of 30; there was general stiffness of the muscles of the forearm and legs, the wrists were flexed and resisted extension, and the position of the fingers corresponded with Kinnicutt's description. The feet were moderately rigid and extended and the toes were flexed. Both these cases were treated with lavage and recovered. Severe cases have been almost uniformly fatal.

Diagnosis.—The presence of tetany, even when undeveloped or in a latent state, may be determined by the application of the classical tests, all of them depending upon hyperirritability of nerve tissue. Trousseau's phenomenon, the first of these tests, consists in making pressure along the nerve trunks, especially those of the upper extremity. The second, known as Erb's phenomenon, depends upon the electrical over-excitability of the nerves, particularly of the upper extremity. It is the galvanic irritability which is striking. A cathodal closure is produced by the feeblest current, .1 milliampere, which easily induces tetany. Anodal closure or anodal opening which in health never produces tetanic spasm, no matter what the strength of the current, readily produces such a reaction in tetany, even when the current is feeble. A third test, known as Chvostek's phenomenon, depends upon the fact that tapping over the facial nerve, at a point about midway between the zygoma and the angle of the mouth, produces a lightning-like contraction of the muscles supplied by this nerve; that is, a contraction is caused at the angle of the mouth, the sides of the nose, and even about the inner canthus of the eyes and the eyebrows.

**Prognosis.**—Cases of the severe type are usually fatal, a result either of the intensity of the spasms or of their long duration. Yet the milder type of cases may recover even after having persisted for months.

Treatment.—Our present incomplete knowledge of gastric tetany suggests that some toxic substance may be present in the stomach contents and that, absorbed into the blood, it leads to a perverted action on the part of the parathyroid bodies and hence induces a disturbance in calcium metabolism. For this reason it would seem wise to practice lavage systematically. However, this course is opposed by many. When there is obstruction, surgical drainage of the stomach is indicated. In addition calcium lactate should be given by mouth, 1 gm. from four to eight times a day; and when the symptoms are urgent the drug should be intravenously employed.

THE SUBGICAL TREATMENT.—The operation that will provide the most perfect drainage appeals to one's reason, especially when it is recalled that 80 per cent of cases medically treated have died. If it were certain that the toxemia did not depend upon a perverted internal secretion of the stomach, surgical drainage should be urged uniformly.

J. H. Cunningham, in reporting a successful case, and in reviewing those reported by others, urges early operation.

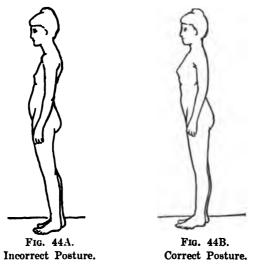
The results show that mild cases recover and severe cases do not.

### CHAPTER XXIV

## GASTROPTOSIS—ENTEROPTOSIS—ABDOMINAL MASSAGE

The older physicians were acquainted with the displacement of various abdominal viscera, yet little importance was attributed to the matter until Glénard called attention to its frequency and gave the name enteroptosis to a morbid abdominal condition which he looked upon as a clinical entity. Further study of the subject, while leading us to a conception somewhat different from that held by Glénard, nevertheless verifies most of his conclu-Virchow announced that a normal position of the abdominal organs in adults was rarely found at autopsy. Stiller regarded the condition, which he spoke of as splanchnoptosis, as marking imperfect development, for he found that it appeared in persons having a special type of conformation, or decubitus, in which a weakened ligamentous apparatus was a prominent feature. was inclined to place the responsibility for the poor development upon a faulty nutrition incident to malposition of the abdominal organs. Extensive observations demonstrated that the condition was much more frequent than had been supposed. Meinert showed that the majority of women suffered from gastroptosis and that it was found in only five per cent of men, a difference which Matthew D. Mann would attribute to the wearing apparel of women. It is not often seen under the tenth year of age, but from this time onward its frequency is progressive and it is most commonly observed in the later years

of life. It was natural to hold tight dressing and corsetwearing as responsible for the preponderance of the deformity in women and, undoubtedly, the continued wearing of apparel that constricts and weighs down the abdomen does contribute greatly to visceral displacement. However, it became evident with increased study of the subject that there was a predisposition to the disease in certain individuals regardless of dress. In these it was found that there was a faulty posture and a laxity of



the abdominal walls, and a consequent lowering of the intra-abdominal pressure, which almost necessitated the downward displacement of the abdominal contents. See Fig. 44. Child-bearing, from the resulting stretching of the walls of the abdomen, was recognized as a factor in producing these displacements and yet it was apparent that enteroptosis occurred with great frequency in spinsters and in comparatively young girls without the contributing effect of parturition or the wearing of close fitting apparel. The fault has been referred to weakness of the abdominal ligamentous supports, and Stiller called

attention to the fact that there was an absence of the tenth costal cartilage in the enteroptotic which he regarded as representing a feature of a widespread defect in the ligamentous and cartilagineous supports. quite evident that, regardless of the influence of these supports, a downward displacement of the viscera is almost inevitable in the presence of over-yielding and flabby abdominal walls, and the lowering of intra-abdominal pressure which this effects. As an illustration of the result of increased surrounding pressure, let us place an apple, a turnip and an onion in a vessel and then fill it with water. Lying at first in the bottom of the vessel, these are floated when the water reaches a certain height: in other words when the increased surrounding pressure lends them sufficient support. Granting that the abdominal viscera may be held up as a result of a greater pressure about them, it was argued that the condition of enteroptosis would become impossible if the intra-abdominal pressure were sufficiently raised. The occurrence of weak abdominal walls in the great majority of cases of enteroptosis and the consequent decrease of intra-abdominal pressure is a relationship that cannot be ignored. Nevertheless, it must be confessed that we at times find that one organ shows a much greater degree of displacement than exists in others, and this may occur in individuals who have no special feebleness in conformation, and who do not belong to the type described by Stiller. The factor of intra-abdominal pressure therefore applies to these individuals in a minor degree.

Experience leads me to conclude that enteroptosis is involved with the general question of faulty physical development. Not only is it necessary to procure a strong abdominal wall, but to an equal extent to form the habitual practice of keeping a correct posture. This includes

not only an erect carriage, but the preservation of the normal spinal curves. The great majority of enteroptotic patients show postural defects that one soon comes to regard as characteristic of the condition. The subject has received enlightening consideration by Goldthwait 1 The subject is shown to be important if somewhat complicated; yet there can be no question as to the need of familiarity with it if the various forms of ptosis are to receive proper preventive and curative treatment. There are two types of spinal deformity described by Goldthwait as productive of visceral displacement. In the first there is an exaggerated dorsolumbar curve and a compensatory exaggeration of the backward lumbar (See Figs. 45 and 46.) This results in shortening the abdominal cavity and in throwing the lower part of the abdomen outward so that it protrudes. At the same time the head and cervical spine bend forward, thus elongating the cervical fascia which normally sustains the diaphragm in position. As a result of the lengthening of this fascia, the dome of the diaphragm descends. Being sustained at its cardiac end by the diaphragm, the stomach necessarily falls with the depression of the former. As a result of these mechanical deficiencies acting conjointly, there comes about ptosis of all the abdominal vis-The abdominal parietes naturally relax with this faulty posture, and the muscles are still further weakened by the outward pressure of the descending viscera, until finally very little support is afforded to the abdominal contents; consequently there is a lowered intra-abdominal pressure. This type, having an exaggerated anterior

<sup>&</sup>lt;sup>1</sup> "The Relation of Posture to Human Efficiency and the Influence of Poise upon the Support and Function of the Viscera," American Orthopedic Association, 1909; also, "The Recognition of Congenital Visceral Ptosis in the Treatment of the Badly Poised and Poorly Nourished Child." Am. Jour. of Orth. Surg., Nov., 1911.

dorsolumbar curve, is frequently observed in children with faulty development and enteroptosis.

Another type of defective spinal posture and poise is seen in those in whom there is an almost complete absence



Fig. 45. — Exaggerated Dorso - Lumbar Curve with Compensatory Exaggeration of Backward Lumbar Curve.



FIG. 46.—CORRECTED POS-TURE.

of the normal dorsolumbar curve with bending forward of the head and cervical spine and a forward droop of the shoulders. (See Fig. 47.) In this condition there also occur slackening of the cervical fascia, relaxation of the abdominal wall and visceral ptosis. In both these types the relations of the viscera are so deranged that a large part of the support supplied by the shelf-like arrangement at the back of the abdominal cavity is lost.



Fig. 47.—Absence of Normal Dorso-Lumbar Curve. (By permission of Dr. Goldthwait.)

The support given to the abdominal viscera by these anatomical ledges or shelves has been studied by a number of anatomists and clinicians, by whom it has been shown that the support which they lend is of material importance. When this is lost, ptosis easily results.

It will thus be seen that gastroptosis is rarely a mere local defect, but is rather the result of a general predisposition, which relates to the posture of the entire frame of the individual. One who attempts to restore efficiency to patients of this kind will find that he faces not only the problems of visceroptosis, but often at the same time flat chest, round shoulders, weak back, iliosacral relaxation, pronation of the ankles, flat foot, retarded circulation, general suboxidation, with a variety of attending symptoms. Dr. Richard R. Smith of Grand Rapids, Michigan, has accumulated a large number of graphic representations showing the conformation of victims of enteroptosis from which he concludes that the condition usually occurs in people of frail type, mostly women, having certain peculiarities in form, such as sloping shoulders, flat chest, prominent abdomen and a remarkable lack of normal dorsolumbar curve. He also shows that a marked change in position of the stomach may occur with change of posture. Having seen troublesome cases of enteroptosis who suffered at the same time from lordosis, I was unprepared for the statement that the absence of a normal dorsolumbar curve is a prevailing characteristic in enteroptosis; however, now that my attention has been directed to the matter, I find that the observation is correct in a goodly number of cases.

Smith agrees with Goldthwait in referring the condition to hereditary and acquired causes, especially the former. Though the tendency appears in childhood, marked ptosis rarely occurs until later life. Radiographic studies show that normally the stomach is high in children and the colon in ideal position with elevated splenic and hepatic flexures. Smith believes that the symptoms so common in enteroptosis result rather from its causes than from the malposition itself. Among those

who have given practical study to gastro-intestinal diseases, there will be a concurrence of opinion that although a general frailty of type, accompanied with impaired nutrition, may account for the development of symptoms, there can be no doubt about the fact that malposition of the abdominal viscera of itself produces varied and important symptoms. Each organ displaced may engender its own peculiar train of symptoms, which, of course, are not invariably present. Curiously enough, a slight displacement may give rise to serious inconvenience while a marked displacement apparently may cause no trouble. These general considerations of visceroptosis have a direct bearing upon the subject of gastroptosis.

### **GASTROPTOSIS**

Holzknecht and Groedel conclude from radiographic studies that the great majority of healthy adults exhibit a condition of moderate enteroptosis, including a low situation of the stomach, and that in an early period of life the stomach tends toward a vertical position. This view must not be accepted in its entirety, for insufficient value is attributed to the effect of gravity in dragging down viscera weighted with bismuth. Meinert concluded that the stomach at birth has the horizontal position, but that it very soon assumes the relatively vertical; a result that follows in the lapse of years from the upright posture. Holzknecht insists that a vertical stomach is not necessarily expressive of ptosis for he found it thus placed in from 15 to 20 per cent of persons who exhibited no signs of ptosis in other organs, a view which has the support of Pfahler and others. Two types of the vertical stomach are described: (1) the so-called "steerhorn" type lies upon the left side of the abdomen with the lower part reaching slightly below the umbilicus; (2)

the so-called "fish-hook" type corresponds with the other except that at about the beginning of the last third it bends rather acutely upwards and to the right so that the pylorus occupies a higher position than in the former type. In the downward displacement of the stomach, it may happen that the descent is most marked toward the antrum pylori, at the pylorus, or the whole organ may descend below its normal position. These facts concerning the position of the stomach as well as its shape were not appreciated until the problem was studied with the aid of the Roentgen rays. (See Radiogram XI.) We now recognize that in life and during its physiological activity the shape and, to some extent, the position of the stomach are not in accord with that which is found post mortem and upon surgical exploration.

Abdominal surgeons have long confirmed the view of physicians as to the frequency of gastroptosis, but previous to the use of the radioscope we did not appreciate the facility with which the stomach changed its relations. Formerly much confusion existed between gastrectasis and gastroptosis, and too much importance was attributed to the position of the lower border of the stomach. The question of gastroptosis is a relative one and cannot be determined merely by the relation existing between the lower border and the umbilicus.

The umbilicus has been selected as a fixed point in the topography of the stomach. When the stomach is in position within normal limits, of normal size and moderately distended, we have expected to find its lower border slightly above the umbilicus. Attention has been called by Haussmann of Germany and by Pron in France to the fact that the position of the umbilicus does not bear a fixed relation to the skeleton nor to the position of the viscera, and that it may be in a position higher or lower

regardless of the height or general conformation of the A transverse line passing horizontally through the umbilicus in the average individual is found to be at about the lower border of the stomach; but when we find the lower border of the stomach passing below this transverse line or above it, before we conclude as to the position of the stomach in its relation to the other internal viscera, we must consider the relative position of the umbilicus. A more reliable guide is that which is based on a line that passes transversely across the abdomen connecting with the ends of the eleventh ribs. But even this mark is not perfectly reliable and, in estimating the size of the stomach or its position, we must be guided by the size and shape of the abdominal cavity and by the relation between the position of the stomach and the other organs.

In the normal individual we find the fundus of the stomach occupying a region which from the standpoint of physical diagnosis is in the thorax. For the most part the stomach is enclosed by the thoracic cage; it reposes well back towards the spine; then bending forward, may be recognized by the resonance to be developed over Traube's space. The cardia is at the left border of the sternum, behind the sixth and seventh costal cartilages. corresponding with the tenth dorsal vertebra. lorus at its superior border corresponds posteriorly with the second, third or fourth lumbar vertebrae and is often partly covered by the liver. Its center lies at the junction of a line drawn down from the right border of the sternum and a horizontal line drawn somewhere above the umbilicus. It is about 3 cm. to the right of the median line when the stomach is in a state of repletion, but with an empty stomach it is usually at the median line or slightly to the left thereof.

In gastrectasis or when the organ is heavy with contents the pylorus may be dragged down and carried to the left. Or when the antrum pylori is dilated and distended, it may bulge so far to the left that the pylorus is concealed in front, as shown by Radiograms IX and XII. The upper two-thirds of the lesser curvature is parallel to the left border of the sternum, thence its lower third bends transversely to the right. The horizontal portion passes at a point about 4 or 5 cm. below the xiphoid cartilage. When not dilated the lower one-third of the stomach during its physiological activity assumes a tubelike form. However, in a state of ptosis the form of the stomach frequently fails to correspond to this anatomical description. This may depend upon dilatation which is so frequently associated with ptosis. In fact, dilatation is a contributing cause to ptosis, owing to the increased weight which appertains to food stagnation. The shape of the stomach may also be modified by adhesions to surrounding organs, a condition which may anchor the stomach in an unnatural position and through subsequent contraction of adhesions it may be drawn downward, thus creating gastroptosis.

Symptoms and Diagnosis.—Gastroptosis is usually unaccompanied by symptoms sufficiently specific to lead one to suspect its presence. This is evident from the fact that though the condition is extremely common, it often goes unrecognized. Hence the statement that gastroptosis per se does not produce symptoms and that the ill-health which accompanies it is the result of very general conditions and peculiarities in development which have led to the gastroptosis. There is complaint of a sense of depression and weight in the abdomen which is relieved by the application of artificial abdominal supports. It is possible that this relief is, at least in part, dependent

upon psychic conditions. Occasionally, but not often, delay in the emptying of the stomach and the symptoms of motor insufficiency are found to depend upon gastroptosis. For the most part, patients suffer from weakness, from gastro-intestinal auto-intoxication and from a variety of sympathetic nervous disturbances believed to be excited by the displacement of abdominal viscera. Glénard called attention to the ease with which the impulse of the abdominal aorta could be palpated in this condition owing to the fact that in the epigastric region the abdominal wall approaches the aorta too closely. Some patients become over-conscious of this pulsation and are relieved when the enteroptosis is corrected.

The downward displacement of the stomach may be recognized by palpation when the stomach is moderately distended. This may be assisted by the conjoint practice of palpation and auscultation. The precise position of the stomach may be shown more strikingly by having recourse to inflation with gas. Four gm. of sodium bicarbonate, 3 gm. of tartaric acid, separately dissolved and taken one after the other give rise to the evolution of sufficient gas to inflate the stomach and render its form and position easily recognizable. About 1½ liters of gas may be expected from this amount of alkali and acid. More or less than this may be required to provide suitable distension. It is well to proceed guardedly. Those accustomed to the use of using a little at a time. the stomach tube are enabled to estimate somewhat closely the lower border of the stomach. It is not wise to distend the stomach with fluid. The radiogram has of late been employed for the final test of gastroptosis. However, it cannot entirely replace the other methods of examination. Indeed it may lead to a wrong inference owing to the displacement which the weight of the bismuth produces, especially in the standing posture.

Treatment.—The most reasonable means of overcoming the condition is by increasing the intra-abdominal pressure. The best methods of accomplishing this are those that are physiologic; that is, those that develop the strength of the abdominal muscles and correct the posture of the patient. A well-directed course in medical gymnastics will accomplish more than any other method of treatment. Prompt relief may be procured temporarily by the application of adhesive straps which support the abdomen, as recommended by Rose. The disadvantages of this method may be avoided and similar results obtained by the wearing of a carefully fitted abdominal bandage or of certain types of corsets which make pressure upwards as well as inwards. The various surgical procedures which look to hold the stomach in a higher position are generally unwarranted. It is only in those rare instances in which definite and distressing symptoms such as follow mechanical obstruction or biloculation, can be shown to depend upon displacement of the stomach that one should consent to an operation. these cases the surgical support of the organ has led to very satisfactory results. It will be seen that the treatment of this condition is mostly hygienic and developmental. These measures, so vitally important to the welfare of many patients, are often omitted merely because they are so general and because they have to be skillfully practiced and long continued. Undoubtedly something can be accomplished by superalimentation in the very lean for the reason that an increase of abdominal fat affords a better support to the abdominal viscera. But this method has limitations, for in an attempt to increase the body weight one often induces in these cases

of enteroptosis food poisoning and intoxication which result from imperfect digestion and faulty elimination. The practice of full feeding, therefore, should be undertaken only in connection with other carefully prescribed hygienic rules, of which gymnastics and open air life make up the larger part.

Lest these recommendations for the upbuilding of systemic strength, the correction of postural defects and the acquiring of efficient abdominal tonus may seem too general, there are added specific directions for gymnastic treatment. For these rules I am indebted to Dr. Kaveleff Mankell.

To begin with, teach the correct standing position, as that gives all the organs the best relative position and mechanically the easiest work, as follows:

- (1) Standing with weight on the balls of the feet; straight knees, without stiffness; not too pronounced anteroposterior curves, whether in cervical, dorsal or lumbar region, nor obliterating of lumbar curve; the head high, chin down, chest raised and abdomen drawn in. To assume this posture requires time and training. (See Fig. 49.)
- (2) Exercise for Abdominal Ptosis and too Pronounced Lumbar Anterior Curve, as in Lordosis.—(See illustration showing lordosis, Figs. 48, 49, page 544.) Stand with lumbar part of the back against a wall or any straight surface. To do this requires flexibility and at first the feet may have to be placed several inches in front of the wall. The head and chest should bend forward in a stooped position. (See Fig. 48.) Gradually as the spine gets more flexible, the feet can be placed nearer to the wall and the chest and head higher, until the patient can touch the wall with the whole spine at one time. (See Fig. 49.)
  - (3) Deep Breathing.—The patient should be lying

flat on the back. Teach to inhale deeply, lifting chest and abdomen at the same time; exhale, lowering both at the same time. It helps the beginner to instruct him to inhale through the nose and to feel the current of air in the roof of the nose, as when inhaling deeply to smell; also to place one hand on the abdomen, the other on





Fig. 48.

Fig. 49.

Figs. 48 and 49.—Exercise for Abdominal Prosis and Too Pronounced Lumbar Anterior Curve. Standing Position.

the chest, and feel the rising and falling parietes. A slow, even, rhythmical movement continued from two to ten minutes is more desirable (for house practice) than a few breaths taken with the utmost effort.

(4) Lying on the back, patient lifts one knee to the

chest and voluntarily draws in abdominal muscles, at the same time breathing in, then back to position. Repeat with other leg, then both together, movements to be taken six to twenty times. The abdominal muscles must be contracted at the same time else the exercise loses in effect, as it can easily be executed with mainly the iliopsoas group. Patients may with the hands press knees close to chest when limit of voluntary movement is reached.

(5) Lying on the back, lift both knees to the chest; then turn both knees to the right side; down, then to the



Fig. 50.

EXERCISE FOR ABDOMINAL PTOSIS AND TOO PRONOUNCED LUMBAR ANTERIOR CURVE, LYING POSITION.

left side; up again in a circular movement, and repeat six to fifteen times, then reverse. The circumduction with one leg at a time can be done by help of the physician.

- (6) Leg Elevation.—Lying on the back upon a plinth, mattress or bed with hands grasping a rod over the head, lift one leg to vertical position. Then bend knees toward the chest as in exercise No. 4, and return to position. Repeat with other leg. Both legs at the same time can be lifted when the muscles become stronger. Repeat four to fifteen times.
  - (7) Sitting on a plinth or hard surface with legs

straight ahead (knees straight) support hands on the same plane. Lift weight of body on the hands and raise the chest high. (See Fig. 51.) Repeat four to ten times.

- (8) Sit in the same position as No. 7, but stretch arms high up and grasp a support.
  - (9) Same position as No. 8, but knees bent.
  - (10) Creeping position, but with forearms supported



Fig. 51.

from elbow to the hand, stretch knees so that the body forms an arch from head to toes. (See Fig. 52.) Then back to kneeling position.

Exercise for patients with obliterated lumbar curve, and generally too pronounced dorsal posterior curve as in kyphosis. (See illustration "A"-page 531 and Fig. 47, p. 535.)

Practice exercises Nos. 3, 4, 5, 6, but with a hard pillow between shoulders, arching body forward, or in Goldthwait position, which increases the flare of the ribs.

No. 8 with a hard pillow between shoulders, arching body forward.

- (11) The patient sitting or standing with support to body by assistance at, or below shoulders, bending body backwards.
- (12) Patient standing, hands straight up, grasping stall bar or other support; the physician draws body forward, his hands behind patient's shoulders.
- (13) Lying, support back of head and heels firmly, arch chest upwards (opisthotonos position).



Fig. 52.

EXERCISE FOR ABDOMINAL Prosis and Too Pronounced Lumbar Anterior Curve.

- (14) Breathing Exercises (Assistive).—Sitting, with chest elevation; physician standing behind patient; patient sitting on a stool without back. Support patient's back with a pillow which rests against the physician who lifts patient's arms and shoulders upon inspiration, compresses chest at end of expiration.
- (15) Patient, half lying, or flat on back; lift chest with hands placed under the patient's shoulders (scapulae); at inspiration, compress chest with voluntary expiration.
  - (16) Teach patient deep breathing in lying, sitting,

standing position, and while walking and counting steps. (17) Lateral Trunk Exercises.—Trunk circumduction; patient sitting astride a high plinth, with knees wide apart and (still better) properly supported; the body should then describe as large a circle as possible for-



Fig. 53.

Exercise for Abdominal Ptosis and Too Pronounced Lumbar Anterior Curve.

ward, to the right side, backward, to the left side, four to fifteen times; then reverse the movement. This can be done with the help of the physician, the patient being passive; or actively, by the patient without the aid of the physician.

- (18) Sideways Bending.—Same position as No. 12, but hands free, patient bending body to the left side, as far as possible, then to the right. Repeat four to fifteen times.
- (19) Trunk Rotation (Twisting).—Same position as No. 17. Turn body as far as possible to the left, then forward (breathing in while doing so), then breathe out, turning to the right, and reverse. Can be done against resistance by physician or alone.
- (20) Postures Which the Patient Should Assume and Continue to Hold from a Few Minutes to Half an Hour.—Trendelenberg's: Patient on back with elevated hips.
  - (21) Goldthwait's with elevated chest; Fig. 51.
  - (22) Goldthwait's with head and face down.
  - (23) Knee chest with abdomen drawn up.
  - (24) Lying on back, the foot of the bed raised 18 inches.

It is easier to follow a general schedule, but selection from the above described manipulations, exercises or positions, or variations from these, undoubtedly would give individual patients a better result. Other exercises may be added, as for instance, shoulder-blade exercises, balance exercises, exercises for ankle pronation or weak feet as may be required in an individual case. Each patient should have spinal exercises that tend to correct any existing faulty position. Yet in every case breathing exercises and cultivation of the habit of deep breathing should be practiced, besides abdominal exercises. knee elevation, leg elevation and lateral trunk movements. Beginning with a few exercises such as the patient can take without fatigue, increasing as the patient is ready is advisable. A wise training in general muscle control is always of advantage, teaching the patients to use just enough force to accomplish habitual movements and not to work with hypertonus of muscles. Thus they

may be taught endurance, rather than severe sporadic efforts, which attain little except fatigue.

These exercises I know from long personal experience will accomplish remarkable results. They must be directed intelligently, and therefore the physician must become reasonably expert. It is not easy to understand the effects of gymnastics until one has himself practiced the art. That we, as a profession, have neglected this important work is evident and every practitioner who has not availed himself of medical gymnastics and mechanical therapeutics is urged to undertake it and to assist in its further application.

# CHAPTER XXV

#### ABDOMINAL MASSAGE

During massage the patient should lie on the back, with hips raised and knees flexed, the plane of the abdomen slightly inclined towards the head. The normal spinal curve should be accommodated with suitable support from cushions.



FIG. 54.—Position for Abdominal Massage in Splanchnoptosis.

- (1) Effleurage.—Deep friction with the palm of the hand over the region of the stomach from left to right toward the pylorus, using a deep, even motion.
- (2) Surface Friction.—With back of the nails of one hand, using a quick, to and fro motion on the skin over the area of the stomach.
- (3) Petrissage.—Deep kneading of the stomach with the finger tips, describing a deep circular motion; the main direction from the left toward the pylorus. One hand may be superimposed upon the other to increase the force.

- (4) Deep Kneading.—Sink ulnar side of hand deeply below the stomach, grasping it with the whole hand, kneading it thoroughly yet without roughness and without causing pain.
- (5) Vibration.—With the hand over the stomach (or during an attempt to lift the stomach), at the same time pushing the contents toward the pylorus. A good mechanical vibrator may be used as a substitute.
- (6) Shaking.—With the hand flat over the region of the stomach.
- (7) Pressure.—Steady and deep, with finger tips of one, or both hands held close against each other, penetrating further during expiration. Between the ensiform cartilage and the umbilicus the solar plexus may be reached, and soothed or stimulated.
- (8) Vibratory Pressure.—In the same manner as in No. 7, except that vibration is added to the movement, making it better tolerated by many patients.
- (9) Percussion over the Stomach.—With finger tips, or ulnar side of the hand, fingers slightly flexed; with elastic wrist movement.

Many different combinations of manipulation and somewhat varied technique may be used.

General abdominal massage often should be added.

For the small intestines the main movements are as follows:

- (1) Put palm of hand transversely over region of small intestines; with a to and fro movement, push intestines from side to side, by changing the pressure from the finger tips to the palms of the hand and not by gliding over the surface.
- (2) Deep kneading with finger tips, making a circular motion, using one hand and perhaps the other also, superimposed.

- (3) Shaking, with hand over same area.
- (4) Vibration.

Colon massage, particularly of the ascending part, is important. This should be directed especially towards emptying the cecum and ascending colon. Intermittent massage should be applied over the transverse and descending portions.

- (1) Effleurage. With the whole palm of the hand, fingers pointing down, a slow steady stroke upward from the pelvic brim on the right side, to the hepatic flexure. The flank may be supported by the other hand, thus exciting counter pressure. While massaging the ascending colon the physician should sit at the patient's right, facing the same way as the patient for in this position a deep, even stroke may be excited conveniently.
- (2) Deep kneading in small circles, as before described, working upwards.
  - (3) Vibrations.
  - (4) Shaking. Finish with effleurage (No. 1).

Follow the transverse colon as well as possible with deep kneading in small circles. Effleurage of the transverse colon is not advisable when there is any difficulty in the emptying of the stomach. In manipulating the descending colon it is a question whether deep stroking downwards does not tend to drag an already depressed intestine further down. It thus seems better to avoid any pushing down movement, practicing only the deep kneading in small circles, taking care to press upward rather than downward and depending for effect rather on the general stimulation and nutrition produced than on the actual propelling of the intestinal content toward the rectum. Massage of the back and deep pressure or vibratory pressure on each side of the spinous processes, one inch out (trying to locate the spinal nerve exits) aids in

the general stimulation; also vibration and percussion with a "concussion hammer" or special apparatus. According to Abrams, concussion over the spinous processes of the first, second and third lumbar vertebra gives a reflex of contraction to the stomach and intestines. It is desirable to learn the precise topography of the viscera, through the aid of radiograms before undertaking a course of mechanical treatment.

### CHAPTER XXVI

### **GASTRITIS**

## GENERAL CONSIDERATIONS

Ten years ago Riegel noted the frequency of the conventional diagnosis of gastritis then so generally made in explanation of dyspeptic symptoms with little regard for the real pathology of the case. The diagnosis of gastritis is not now so generally made without just reason as at that time, but even now it is too often made without verification by close study of cases. It is commonly supposed that a diagnosis of gastritis is an easy matter, merely from the clinical history of the case. This idea is wrong. Gastritis is the most frequent of the structural diseases of the stomach, yet at times a positive diagnosis is made with more difficulty than in the case of the other less common structural diseases. This difficulty is due to the fact that the symptoms of gastritis are merely the symptoms of dyspepsia and, as we have seen, these symptoms are produced most frequently from functional nervous disturbances, from toxic conditions and from various other organic diseases of the stomach. not be inferred from this that gastritis may not associate itself with the several conditions mentioned. As already stated, the various agencies that lead to functional nervous disturbances may also predispose a patient to the action of infection.

Thus we know that inflammation makes up a part of the history of cancer as well as of ulcer of the stomach; nevertheless, the symptom-complex attending cancer and ulcer in large part does not depend upon accompanying gastritis and may be present when there is no gastritis. This statement relating to ulcer and cancer is still more applicable to the neuroses and functional disturbances. How, then, is gastritis to be recognized if its symptoms are nearly identical with those which are associated with functional disease or with other organic diseases? The answer to this is that the diagnosis usually cannot be made from symptomatology alone. We may assume gastritis to be present when gastric symptoms develop in connection with the acute infections, or after the ingestion of highly irritating substances, basing our diagnosis in these instances not so much upon the stomach symptoms as upon the recognized predisposition of the infections to set up gastritis. This is a matter of inference rather than of actual diagnosis. Excluding presumptive evidence of this kind, a positive diagnosis of gastritis is reached only by examination of the stomach contents removed through the stomach tube. Such is the difficulty of the problem that even with accurate laboratory methods it is not always possible to recognize the Confusion arises because previous to the development of inflammatory changes in the gastric mucosa sufficient to produce characteristic findings in the stomach contents, there will have developed dyspeptic symptoms which may depend upon other causes. We may guess that we are dealing with the onset of gastritis because of the abruptness of the attack or because of the absence of special reasons for the appearance of a functional disturbance. This method of reasoning may serve us lamely when the disease arises in a previously healthy person, but how shall we recognize the gastritis which develops in a patient who is already suffering from dyspeptic symptoms of nervous or reflex nature? Under these circumstances, the diagnosis cannot be made until such structural changes occur in the gastric mucosa as to produce a characteristic change in the stomach contents, evidencing thus the inflammatory process. Care and time are required for investigating before we make a positive diagnosis of gastritis.

Classification.—Attempts have been made to classify gastritis on anatomical grounds. Thus, one speaks of catarrhal gastritis, or gastric catarrh; mucous gastritis. in which inflammation attacks the more superficial structures of the mucosa; parenchymatous gastritis, in which the inflammation extends through the ducts and into the glandular parenchyma of the organ; and interstitial gastritis, which particularly involves the connective tissue framework of the mucosa. This classification is simple but not altogether satisfactory because the mucous membrane of the stomach is unlike the other mucous membranes: it is something more than the mucous membrane. True there are distributed a vast number of muciparous glands, especially on the exposed portions of the gastric mucosa, yet the greater part of this structure does not have a superficial exposure but dips down through countless ducts to the deep glandular parenchyma which has a specific secretion and only a slight secretion of mucus. It is proper to look upon the stomach as being lined with a glandular parenchyma as much as with a mucous mem-It is therefore difficult to speak of a superficial gastric catarrh. To what extent does such an inflammation involve the ducts and their glands? This question is not easily answered, for we possess very little histological knowledge of the milder types of gastritis. As is well known, after death the gastric mucosa undergoes remarkable changes through the immediate digestive action of the contained ferments. This obscures the evidences of inflammation. Our best knowledge of the nature of gastritis is gathered from examination of the stomach lining through accidental openings extending through the abdominal parietes or through artificial openings, as after gastrostomy. No study of this kind has surpassed the brilliant and long-continued observations made by the American, Beaumont, in the case of Alexis St. Martin. These painstaking observations in the first place demonstrated that a gastritis of considerable extent might exist without the development of any symptoms whatever; they also showed that the symptoms might be only in a slight degree local, lassitude, anorexia and dryness of the mouth being complained of without any special gastric distress. Beaumont also demonstrated that the lesions of gastritis might be localized, the greater part of the gastric mucosa escaping involvement. It was further observed that the local engorgement and tumidity that was found on some occasions, disappeared very quickly, leaving no vestiges of inflammation. It is hard to believe that these areas of localized gastritis were limited to the more superficial parts of the mucosa. We have grounds for believing that the glandular tissue is involved in supposedly simple cases of so-called gastric catarrh, and therefore we must come to the conclusion that a simple catarrhal inflammation limited to the superficial portion of the gastric mucosa exists only hypothetically, unsubstantiated by observations, and that actually this may not exist as a definite type of gastritis. We find the same difficulty in the attempt to distinguish between the parenchymatous gastritis and interstitial gastritis. How are we to know that the parenchyma alone is involved and that the connective tissue of the mucosa escapes?

Such pathological material as we command indicates that a sharp distinction between the parenchymatous and

the interstitial types does not exist, but that in one case parenchymatous changes predominate and in another interstitial. In all cases the two elements are more or less simultaneously involved. The situation may be likened to that which exists in interstitial nephritis as compared with parenchymatous nephritis; with both these there is a diffuse nephritis.

Etiological classification of gastritis has been tried and it presents certain advantages. Thus, toxic gastritis pertains to cases which result from the swallowing of the corrosives and mineral poisons. An irritative gastritis may follow the ingestion of coarse foods or of foods in excessive quantities. A secondary or sympathetic gastritis is one which develops in the course of some other disease, as for instance, scarlatina, diphtheria or nephritis.

Everyone concedes that the subject may be divided into acute and chronic gastritis. To acute gastritis belong those cases which, regardless of cause and of the part of the structure involved, are temporary in their course. To chronic gastritis belong those long continued cases which show little tendency towards spontaneous recovery.

In this connection the word acute might well be supplanted by the word temporary. Acute commonly refers to the intensity and character of an affection rather than to its duration.

For practical purposes it is convenient to recognize and describe acute catarrhal gastritis, acute toxic gastritis, acute phlegmonous gastritis, chronic catarrhal gastritis, sclerotic gastritis and alcoholic gastritis.

## **ACUTE CATARRHAL GASTRITIS**

The term is here applied to the various forms of acute inflammation of the gastric mucosa from numerous causes, regardless of the anatomical structures involved.

**Etiology.**—There exists a predisposition to gastritis in certain individuals. In these a local inflammatory reaction is excited in the gastric mucous membrane from comparatively slight cause. The analog of this is found in the frequent occurrence of inflammation of the bronchial tubes or the upper air passages in certain individuals. There is apparently an inherited weakness of the structure, and it would appear that this tendency exists in certain families, in all members of which a weakness of the digestive apparatus is noticeable. In addition to the inherited predisposition to inflammation, the gastric mucosa may lose its immunity to one or more groups of pathogenic microörganisms. This loss of resistance may be very temporary or long continued. Sensitiveness of the gastric mucosa may make the digestion of coarse or carelessly prepared foods extremely difficult; and, if their ingestion is continued, irritation and inflammatory reaction may result. In some individuals the stomach is unable to digest foods in which fermentation has begun. Gastritis is excited by the ingestion of foods that are not perfectly fresh, as for instance milk improperly cared for, fish too long out of water, "high" game and meats which are poorly preserved by the use of antiseptics. Though people may experience no unpleasant results from eating various sausages and canned meats or fish, with or without the presence of chemical preservatives, others invariably show symptoms of gastric irritation which mark the beginning of inflammation. The eating of certain fish, Spanish mackerel, lobsters, crabs, mussels and other shell fish is especially taxing to these individuals. syncrasy, which probably depends upon the principle of anaphylaxis, renders certain substances unwholesome and even intolerable to certain persons. While this is most frequently the case with certain animal foods, such as

fish, particularly lobsters and shell fish, veal or "high" game, old cheese, buttermilk and eggs, it is by no means limited to these substances. Strawberries, raspberries and certain green vegetables and mushrooms are highly deleterious to an occasional individual. Gastritis may be excited by iced drinks, by excessively hot drinks or by foods over-seasoned with highly stimulating condiments, such as mustard, capsicum or green peppers and the various meat sauces.

The infections excite gastritis both directly and indirectly.

Thrush (from the growth of schizomycetes) may extend and primary mycosis of the stomach may develop as a part of the general infection.

Dieulafov has studied and described some remarkable cases of pneumococcus infection of the stomach with resulting gastritis and ulceration. Gastritis also makes up a part of the clinical history of a large number of the general infections; scarlatina, small-pox and other exanthemata, diphtheria, typhoid fever, anthrax, tuberculosis, influenza, etc. The local expression of a general infection may be especially active in certain parts whereas others are relatively immune: for instance, the pneumococcus may involve the lungs, the genito-urinary tract, the meninges of the brain and cord or the sero-fibrous structures of the joints and the gastric mucosa. The early fatality of certain cases of pulmonary tuberculosis is determined by the innutrition which results from an accompanying gastritis. Gastritis is a frequent complication of uremia. it is a common result of alcoholism, constituting the wellknown "alcoholic gastritis."

Morbid Anatomy.—The gastric mucosa is reddened and swollen. There is an exaggerated secretion of mucus which may be found as a film covering deeply reddened areas. Carillary thrombosis, round-cell infiltration and connective-tissue hyperplasia may develop with varying degrees of intensity. Hemorrhagic points are found and often superficial erosion, hemorrhagic and otherwise. In gastritis of the infections, the process may extend into the submucosa, resulting in loss of substance and true ulceration. Such are the cases of pneumococcus gastritis described by Dieulafoy. (See Fig. 55.)

Clinical Course and Symptoms.—Acute catarrhal gastritis sometimes gives rise to constitutional disturbances with little evidence of local trouble. The tongue is broad, large and indented by the teeth; it shows a thick, mud-

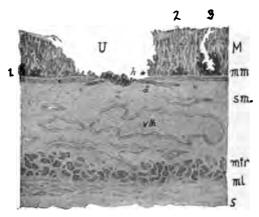


FIG. 55.—SECTION THROUGH THE WALL OF THE STOMACH AT THE SEAT OF SIMPLE ULCER WHICH WAS FORMED AT THE EXPENSE OF THE MUCOSA; M, ALSO OF THE MUSCULARIS MUCOSA, mm; A SMALL SUBMUCOUS ARTERY WAS RUPTURED AT THE POINT h. FROM THIS POINT PROCEEDED A FATAL HEMORRHAGE. v th, thrombosed vein; sm, submucosa; mtr and ml, muscular tunic; s, serous coat; 1, 2, 3, miliary abscesses situated in the depth of the mucosa.

(Plate from Text Book of Medicine, Dieulafoy.)

colored coating, and sticky secretions are present in the mouth. The odor of the breath is disagreeable, the patient shows evidence of auto-intoxication, such as languor, mental irritability, general depression and, at times, a febrile reaction. Fever is not a necessary accompaniment

and should not be taken as the gauge of the severity of the attack. It is doubtless always present in case of deepseated inflammation and infection but it also occasionally occurs in mild cases. To what extent the fever depends upon the gastritis is not known. It may be that the fever is the expression of a systemic condition which accompanies the gastritis and of which the latter is a local manifestation. At any rate, as shown by Beaumont and others, fever may be absent when there is present a considerable degree of inflammation of the gastric mucosa. To illustrate this it is best to quote Beaumont's own words: "Inner membrane of the stomach usually morbid—the efythematous appearance more extensive, and spots more livid than usual; from the surface of some of which exuded small drops of grumous blood—the mucous covering, thicker than common, and the gastric secretions much more vitiated. The gastric fluids extracted this morning were mixed with a large proportion of thick ropy mucus, and considerable mucopurulent matter, slightly tinged with blood, resembling the discharge from the bowels in some cases of chronic dysentery. Notwithstanding this diseased appearance of the stomach, no very essential aberration of its function was manifested. Martin complains of no symptoms indicating general derangement of the system, except an uneasy sensation, and a tenderness at the pit of the stomach, and some vertigo, with dizziness, and yellowness of the vision, on stooping down and rising again; has a thin, yellowish brown coat on his tongue, and his countenance is rather sallow, pulse uniform and regular; appetite good; rests quietly and sleeps as well as usual." 1

Acute catarrhal gastritis often extends into the duo-

<sup>&</sup>lt;sup>1</sup> William Beaumont: "Experiments and Observation on the Gastric Juice and the Physiology of Digestion," 1833, pp. 237-8.

denum giving rise to hepatic congestion, diarrhea and other symptoms referable to the lower digestive tract.

Local Manifestations.—The symptons of acute catarrhal gastritis especially attributable to local conditions are loss of appetite with disturbances of the senses of smell and taste, nausea and at times vomit-After the stomach has been emptied of poorly digested food, the vomiting of a watery mucus, sometimes bile-stained and occasionally showing streaks of blood and fibrinous flakes may occur. In rare instances in which there is marked loss of substance of the gastric mucosa there may be hematemesis. There is usually a sense of fullness with tenderness over the epigastrium; there is motor irritability as shown by eructations and regurgitations. With the decline of the attack atony may develop and give rise to dyspeptic symptoms which may continue after the gastritis, so far as one can judge, has disappeared.

Sometimes, but not always, the stomach is especially resentful toward the introduction of the stomach tube.

Stomach Contents.—The material obtained by lavage shows that digestion is interrupted, and there are usually evidences of fermentation. There is a disagreeable odor to the stomach contents characteristic of fatty and acetic acids. In most cases there is a marked decrease or temporary absence of free hydrochloric acid and perhaps even of combined chlorids. This indicates that the secretion of rennin and pepsin is correspondingly decreased. Mixed with the undigested food is a large amount of mucus, much of it in flakes and strings, perhaps with groups of characteristic cylindrical epithelial cells attached. An unusual number of bacteria are present, leucocytes and red blood corpuscles. Occasionally there are seen fragments of degenerated tissue.

Diagnosis.—One may recognize gastritis from the history and from the nature of the gastric contents. be differentiated from functional disturbances by the history of the case, including the exciting causes, the constitutional disturbance, the gastric derangement and the chemical, macroscopic and microscopic characteristics of the stomach contents. From gastric ulcer it may be differentiated by the facts that in ulcer the digestive processes are usually active, there is lack of the constitutional disturbances and the gastric contents are very dissimilar. Most difficulty will be found in distinguishing between gastritis and cancer. The diseases often co-exist. When acute gastritis develops in a case of gastric cancer it can be recognized only by the temporary change in the symptoms and stomach contents. In pyloric stenosis, either benign or cancerous, there is occasionally acute gastritis, the immediate cause of which is usually an error in diet.

Case No. XXVII.—Pyloric Stenosis, Gastrectasia, Accidental Gastritis, Serious Result, Ultimate Recovery.—I was preparing for operation a patient who had a very advanced case of stenosis resulting from contraction of the cicatrix of a chronic ulcer. Attempt was made to nourish her on white of eggs, since her condition indicated highly nutritious and concentrated food. The patient's insistence that eggs did not agree with her was believed to be but a fancy. She was given during the first twenty-four hours the white of four eggs. Following this there developed a very acute catarrhal gastritis with pain, distress and incessant vomiting of mucus somewhat bloodstreaked. The secretion of hydrochloric acid which hitherto had been at the high mark of 90 to 110 entirely disappeared, but organic acids were found present. The microscopic appearance of the stomach contents was characteristic of gastritis. The patient suffered from moderate fever and from general depression that threatened her life. The attack continued for a week, after which the secretions returned and the gastritis entirely disappeared. The patient was able to take other foods and became sufficiently nourished to undergo operation. Recovery was complete.

Treatment.—Acute catarrhal gastritis, uncomplicated, makes a spontaneous recovery in a comparatively short time, provided the stomach is given rest. It is rarely necessary to practice lavage, although in patients who take the tube easily washing out the stomach and applying through the tube a soothing bismuth mixture produces great comfort and shortens the attack. In most cases it is desirable to clear the alimentary tract by a large dose of calomel, .30 gm. (gr. 5) with an equal amount of sodium bicarbonate, following this six hours later by a draught of Abilena or Hunyadi János water, or by sodium or magnesium sulphate well diluted. Following this. small doses of calomel, .00065 gm. (gr. 1/100), triturated with bismuth subnitrate or subgallate, .30 gm. (gr. 5), and given every hour will greatly advance the patient's comfort; or this alone may suffice with patients for whom a preliminary large dose of calomel seems inadvisable. Equal parts of the milk of magnesium and the milk of bismuth, given frequently, are indicated, and in cases in which there is comparatively little gastric irritability and in which the intestine is especially involved, prompt benefit is usually produced by the following:

Or the following prescription, in case of nausea, may be more suitable:

₽		
	Phenolis	0.25—m. iv
	Cerii oxalatis	
	Aquae chloroformi	15.00—3iv
	Mucil. tragacanthae	30.00—3i
	Aquae destillataeq.s. ad.	60.00—3ii
7.F		

Sig.—Of this a teaspoonful, diluted, should be given every two hours.

An antacid sedative may be required, in which case the following mixture is advised:

₿		
	Bismuthi subgallatae	2.60—gr. xl
	Sodii bicarbonatis	2.60—gr. xl
	Pulvis tragacanthae	1.50—gr. xxiv
	Aquae carui	60.00—3ii
M.	•	
Sig.	Of this take a dessertspoonful, p.r.n.	

When there is local tenderness and distress, a poultice of flaxseed or of flaxseed meal, 1 part, and bran, 2 parts should be applied over the epigastrium. The latter forms a poultice lighter than does flaxseed alone. An excellent poultice for application over the epigastrium is made of boiled potatoes freshly mashed. A full warm bath and rest in bed for a short time is desirable, although in mild cases the patient may be allowed to be about the room. Of all the measures of treatment mentioned the most important is rest of the stomach by fasting. There should be absolute fasting for the first day or two until there is a desire for food and until the disappearance of local sensitiveness. Meantime demulsive or soothing drinks such as toast water, a weak infusion of poppy heads or barley water may be allowed. Alimentation at first should be limited to very thin gruel or gelatine water. and the diet should not be increased until it is manifest that these delicate foods produce no local disturbance. Then the diet may be gradually increased although animal proteids should be withheld until the gastritis has completely disappeared. A full diet should not be allowed immediately upon desire for it, but should be delayed until it is believed that the function of the stomach, including its proper motility, has asserted itself.

## TOXIC GASTRITIS

Cases suffering destruction of the gastric mucosa as the result of the ingestion of mineral acids, the caustic alkalies and other corrosives are not included under this title, but only the minor structural changes resulting from taking these or similar substances in small or greatly diluted amounts. The clinical manifestations resemble those of simple acute catarrhal gastritis but are usually more prolonged. The treatment therefore must be modified accordingly; that is to say, there should be a longer period of fasting and greater precaution taken in resuming and increasing alimentation. At a comparatively early stage demulsive drinks such as have been described are not only admissible but desirable. Except in the case of poisoning by phosphorus and other substances that give rise to marked degeneration of the glandular structures, these cases progress favorably, though more slowly than would gastritis from other causes.

## **ACUTE PHLEGMONOUS GASTRITIS**

Acute phlegmonous gastritis, or interstitial suppurative inflammation of the stomach, although long known, is infrequently encountered and often unrecognized. The disease consists of a pyogenic infection beginning in the deeper part of the mucosa from which it may extend outward through the muscularis and the outer coat of the stomach to the peritoneum, giving rise to peritonitis or

to infection of organs in the vicinity of the stomach. Two types of the disease are recognized. In the first or diffuse type, the mucosa is extensively involved and infection extends with a rapidity which depends upon the virulence of the organism and the resistance of the patient. The second type of the disease appears to be the result of circumscribed infection of the stomach and is characterized by the formation of an abscess or of abscesses. The greater portion of the gastric walls are practically undisturbed.

Etiology.—Little that is positive is known of the etiology of this disease. The mode of invasion leads to the belief that in the diffuse type the infection finds entrance through the gastric mucosa. Just why the stomach becomes unable in certain cases to resist pathogenic organisms is a matter of speculation. From the diseased tissues there have been recovered the staphylococcus, streptococcus, diplococcus, proteus and colon bacillus. Any of these organisms may, and often do, enter the stomach without development of inflammation. It is probable that there is some lesion in the mucosa through which the microbes find entrance, and it is also probable that the individual possesses at the time a lowered immunity to pyogenic bacteria. The circumscribed type of the disease has been known to occur in puerperal and other forms of septicemia. In these cases the formation of the abscess in the stomach is undoubtedly a secondary manifestation, the result of septic embolism.

Morbid Anatomy.—In the diffuse type of acute phlegmonous gastritis there may be purulent infiltration of the mucosa and submucosa in definite areas or there may be a widespread inflammation involving practically the entire mucous membrane. Many times the muscularis escapes. The extent of involvement depends upon the severity and duration of the case and these are controlled largely by the virulence of the infection and the resistance of the patient. The purulent infiltration sometimes extends to the serosa, giving rise to great thickening of the walls of the stomach and to septic peritonitis. It may even pass the diaphragm and invade the pleural cavities. In some of the cases in which abscesses do not occur pus may be squeezed out of the tissues which are swollen and deeply injected as the result of purulent infiltration. There are often multiple miliary abscesses. One of the best descriptions of the diffuse form of phlegmonous gastritis is by Kinnicutt.2 The submucosa in the case which he describes was enormously thickened and even in the hardened sections was in some places at least three quarters of an inch thick. The muscle fibers were swollen and distorted and the subserous layer was irregular, thickened and infiltrated with round cells. A streptococcus was the infecting organism. In some cases the submucosa is necrotic and there are superficial ulcers, probably the result of sloughing. In the abscess type of the disease, there is the ordinary pus cavity usually located in the submucosa or beneath it. The walls of the abscess cavity are indurated and the surrounding tissues infiltrated with round cells. These abscesses may open into the stomach or into the peritoneal cavity.

Clinical Course and Symptoms.—There is at times so much vagueness in the symptoms that the disease passes unrecognized. Previous to the appearance of symptoms of peritonitis the patient shows ordinary manifestations of acute infection and septicemia, with great variation in temperature, which in some patients does not rise above 101 but in others is much elevated, and the pulse varies correspondingly. It may be said that there are no typical

<sup>&</sup>lt;sup>2</sup> Trans. Assoc. Am. Phys., 1901.

symptoms. Vomiting occurs early, then it ceases usually for a time, to begin once more with the onset of peritonitis. An excellent description of a case is given by Wagner.<sup>3</sup> An alcoholic, aged 30, had sharply localized pain and tenderness in the epigastric region, anorexia, frequent vomiting of bile-stained fluid, a furred tongue and great thirst. The pulse was full and strong; the temperature was 101° F. After a week's illness there appeared over the entire body excepting the face, welldefined large and small patches of intense erythema. These were present in the mucosa of the mouth and pharynx. A friction sound was heard over the stomach. Pain was diffused over the entire abdomen. There was tympanites and the general appearance of peritonitis. The patient died from collapse on the tenth day of illness. At autopsy all parts of the stomach were found involved. There was a fibrinopurulent peritonitis and pleuritis. The stomach was not perforated.

Robertson reports a case which followed chronic ulcer of the stomach. In some cases there is vomiting of pus. This is believed to occur more often in the abscess-forming type of the disease.

Prognosis.—The disease is characterized by fatal termination in the great majority of cases. Recovery is more often seen in the abscess type than in the diffuse infiltrating. However, cases of recovery after illness of several weeks are sometimes reported in the diffuse, infiltrating type. Death may be expected at about the end of the first week.

Treatment.—The most rational treatment appears to be that by vaccines and specific sera. Other than this the treatment is limited to that of general septicemia. Lavage has been recommended, but is unsuitable. Some ex-

<sup>&</sup>lt;sup>8</sup> Canadian Pract. and Rev., Jan., 1907.

ceptional cases are found to be amenable to surgical drainage.

## CHRONIC CATARRHAL GASTRITIS

In the remarks introductory to the subject of gastritis it was stated that in the past nearly all dyspeptic symptoms were attributed to inflammation, and that this acceptance of the rôle of gastritis continues although in a less degree even to the present time. The misconception arises most often in connection with chronic gastritis. Chronic gastric catarrh and chronic gastritis are still very familiar terms for explanation of dyspeptic and abdominal symptoms which often are in reality merely sympathetic manifestations of constitutional troubles. Chronic gastritis is not a rare disease, but its occurrence is far less frequent than might be inferred from current clinical reports. The prevailing idea as to the frequency of chronic gastritis has not established itself without reason, for diagnosis may sometimes be very difficult. Then too, in some fairly modern studies there has been found an inflammatory basis for most of the chemical disturbances of the stomach, and these disturbances of secretion have been dwelt upon overmuch as a cause for stomach symptoms. In view of these facts it is not strange that some confusion exists as to the frequency of chronic gastritis and its importance in relation to digestive symptoms. Understanding of the subject of chronic catarrhal gastritis has been made more difficult through the attempts which have been made to classify it. It is here especially that discussions have arisen as to parenchymatous gastritis, hyperplastic gastritis, interstitial gastritis, and mixed types.

Morbid Anatomy.—Histologically a pure type of parenchymatous gastritis is rare; almost always there is some

involvement of the interstitial structure. In certain cases there is hyperplasia of the glandular tissue occurring in groups of cells only. Some authors describe a widespread increase of parenchymatous tissue which occurs particularly towards the lower end of the stomach and which is found in those cases that early give rise to an excessive secretion and usually to hyperchlorhydria. These are the "acid gastritis" cases of Boas and of Hay-These cases which show primarily hyperplasia of the glandular cells show also connective tissue increase; this increase in some regions causes glandular atrophy. In some areas, although connective-tissue proliferation may be slight, there is nevertheless a thinning of the glandular structures. These cases are spoken of as mixed types, but it seems to me that all cases of so-called parenchymatous gastritis are to a certain degree mixed types. It is apparently a question of degree and no sharply drawn distinction can be made between the parenchymatous and the mixed types of gastritis.

There is still more difficulty in dealing with so-called interstitial gastritis. We find cases in which there is marked increase of interstitial tissue, constituting what might be called a cirrhosis of the stomach. It corresponds with interstitial changes in the kidney, liver, pancreas, etc. We are as yet in an uncertain state of mind as to the nature and pathology of cirrhosis in these various organs. For instance in cirrhosis of the liver there is reason to believe that the overgrowth of connective tissue is to some extent a replacement process and that atrophy of the parenchyma antedates the overgrowth or ingrowth of connective tissue. It is probable that the same principle holds true in so-called interstitial gastritis. After all, the matter is of small clinical importance. Of greater moment is the fact that where there

is long-continued catarrhal gastritis the mucus-secreting tissues become dominant. The mucous glands seem to replace the specific glands, and in all cases the term mucous membrane more properly applies to the lining of the stomach than is true of the normal mucosa.

A type of so-called gastritis in which there occur, often for unknown reasons, degeneration and atrophy of the specific glandular cells with disappearance of secretion yet without definite inflammation, has been called by Ewald anadenia gastrica. It is the cause of certain cases of achylia gastrica. The term "anadenia gastrica" relates to the terminal stages of chronic gastritis, to which the term phthisis mucosa is also applied. According to Schmidt, in this degeneration of the gastric parenchyma there develops a form of epithelium which resembles the epithelium of the intestine and which is never found in the normal stomach. These cells he described as cylindrical with definite margins; they are closed above and alternate with goblet cells. They are found covering areas of the mucosa from which have disappeared the normal glandular epithelium. These peculiar cells have the power to secrete mucus in great abundance. According to Schmidt, this explains the absence of normal gastric juice in advanced cases of chronic gastritis and the constant presence of large amounts of mucus. In severe cases of chronic gastritis degeneration may be found in the muscle fibers. For this reason there is a loss of tonicity and contractility, and the occurrence of atony, motor insufficiency, and frequently, dilatation.

Stenosing Gastritis.—This condition, described by Boas, is accompanied by hypertrophy of muscle fibers, giving rise to a narrowing of the pyloric orifice. This may occur without change in the mucosa, although there often is a thickening of the mucosa and submucosa. It

should be noted that these changes are not limited to the pylorus. Kurt von Sury in a study of this subject virtually confirms the conclusions reached by Boas and looks upon this as a disease sui generis, characterized by chronic inflammatory hyperplasia of connective tissue with contraction especially marked in the submucosa and subserosa. However, he finds that the glands of the mucosa ultimately disappear and that the supporting membrane becomes infiltrated. The walls of the entire stomach may contract, the process not being confined to the pyloric region.

Varices of the stomach with hyperemia as a result of hepatic cirrhosis are not very uncommon. Bonaglia, in 19 cases of gastric varix, found 9 following cirrhosis, 1 following syphilis of the liver and 9 in which no liver disease was demonstrated. Chronic hyperemia exaggerates the secretion of the muciparous glands. The presence of excessive mucus in catarrhal gastritis represents an effort on the part of nature to protect the inflamed surfaces. This benign tendency sometimes becomes an embarrassment. Too little attention has been given to the mucus-secreting glands of the stomach in health and in dis-Schultz, Kaufmann, Zweig and others have called attention to the importance of mucus as a normal source of protection to the mucosa and they give reasons for believing that there occurs a deficiency of this secretion in certain individuals who are thereby predisposed to gastritis, ulcer and cancer. Also it is held that when normal mucus is lacking there is increased irritability of the stomach which induces over-secretion of gastric juice.

There is a rare form of inflammation of the stomach that cannot be classified with chronic catarrhal gastritis. The disease involves first the serous coat of the stomach

<sup>4</sup> Verd. Krank., Feb., 1907.

and may be but a part of a general multiple serositis. The outer coat of the stomach becomes thickened and contracted which renders the organ small. Neither the secretion of the stomach nor its digestive power are affected except in proportion to the decreased capacity.



FIG. 56.—POLYPOID GASTRITIS. (Museum, University of Buffalo.)

CIRRHOTIC GASTRITIS—CIRRHOSIS VENTRICULI.—In one type chronic inflammatory changes involve not only the mucosa, but the muscularis; and in another type they involve the serosa and muscularis; they give rise to marked

interstitial hyperplasia and more or less complete transformation of the secreting layer, followed by such contraction of the gastric walls that the stomach becomes Under these conditions its containing power may not be more than 100 or 200 c.c. The stomach retreats behind the costal border and is not palpable. A small amount of food fills the stomach and consequently food is regurgitated unchanged in a manner which suggests spasm at the cardia. Sometimes associated with this process is a polypoid degeneration of the mucosa, the socalled polypoid gastritis. When this occurs it is frequently located at the cardiac extremity and fundus. A polypus growing in this vicinity may become engaged in the cardiac opening. Polypi growing from the lower end of the esophagus seem to be of different nature from these, but the symptoms are sometimes analogous and in either spasm of the cardia may result.

Extensive submucous fibroid proliferation of inflammatory origin, may be found at the pyloric end of the stomach, where it gives rise to a palpable tumor that resembles carcinoma. Œttinger <sup>5</sup> described a case which occasioned obstruction of the pylorus and produced the usual results upon gastric motility.

Etiology.—Chronic catarrhal gastritis is often simply the continuation of acute gastritis in which the original etiologic factors remain active. Much is said as to the evil effects of hasty eating and imperfect mastication in producing inflammation of the stomach, but these causes alone are generally insufficient. If, when the stomach is for any reason depressed, too much effort is required of it, it responds with dyspeptic symptoms but these are not usually the expression of a gastritis. Whenever gastritis actually exists the trouble is enhanced and the symptoms

<sup>&</sup>lt;sup>5</sup> La Sem. Méd., May 7, 1902.

become more evident upon eating hastily and heartily and masticating poorly. It may be said, then, that bad habits of eating put the stomach at a disadvantage and may lead to the appearance of dyspeptic symptoms; and, when gastritis is already present, this will be aggravated. The habitual use of unwholesome, decomposing, irritating or toxic foods will give rise to chronic gastritis. The most familiar instance of this is seen in the so-called alcoholic gastritis, but it must not be supposed that this type of the disease is merely the result of the local effect of alcohol.

We know that it is rather through the deleterious effects of alcohol upon metabolism, upon the nervous system, the liver and other organs, that the gastric mucosa loses its resisting power and becomes an easy prey to other exciting causes of gastritis. Of course, the local effect of alcohol contributes by inducing congestion and irritation. Direct infection of the stomach may result from disease of the nasopharynx or mouth, or from sputa swallowed in cases of pulmonary disease. Anything which gives rise to prolonged congestion of the stomach is instrumental in arousing therein an inflammatory reaction and may be called a predisposing cause of gastritis. It is therefore not surprising that we find catarrhal gastritis so commonly the pathological companion of portal obstruction or of general venous stasis, the result of pulmonary or cardiac disease. In these instances the congested stomach secretes a diminished or deficient gastric juice and is less resisting to bacteria and other exciting causes, such as injudicious alimentation. Chronic gastritis is almost an invariable result of uremia in which the stomach is irritated directly and in which there is also an indirect predisposition to gastritis. The direct irritation is due to the vicarious elimination through the mucosa into the stomach of urea and toxic matter that should be discharged through the kidneys. The predisposition is caused by anemia, edema and other factors in the lowered resistance of nephritis. Other chronic diseases lead to gastritis, sometimes through innutrition and sometimes through auto-intoxication, as for instance, scorbutus, diabetes mellitus, leukemia, chronic anemia, tuberculosis and syphilis.

Clinical Course and Symptoms.—The symptoms of chronic dyspepsia are those of chronic gastritis. It is the old story of capricious appetite, anorexia, periods of nausea, slight or severe, and vomiting which is sometimes periodical in its appearance. There is a sensation of fullness and weight in the epigastrium, of distress, burning, or soreness, and very rarely of pain. Motor irritability occurs with eructations of gas, whether this be present in normal amount or whether abundantly developed as a result of fermentation. There may be regurgitation of food or of gastric juice, giving rise to the well-known symptoms of water-brash or heart-burn. These familiar symptoms of chronic gastritis vary in intensity from day to day or week to week, according to the diet taken and the consequent effort required of the diseased stomach, or according to the general fatigue, nervous depression and increased intoxications which are the penalties of ill-advised strain or the accidents of concurrent disease. Patients become whimsical as to diet and habits of life. For a time they limit themselves to particular varieties of food and to certain hours for eating, believing that they have thus found a panacea. However, each plan is soon discarded, another adopted and in its turn abandoned. Such patients are heard to attribute this morning's distress to the soup that was taken at last night's dinner or to the orange taken at the preceding breakfast.

Great inconsistency is displayed and numberless domestic remedies are tried for their supposed effects in overcoming "gas" or in preventing "sour stomach." There are certain fairly constant systemic disturbances that belong to chronic gastritis, and one of the most important of these is insomnia or disturbed sleep. Distressing dreams, frequent waking or arousing with a sense of fear are conditions frequently occasioned by chronic gastric ir-Many patients are disturbed by rapid heart action. Sometimes there is a real tachycardia. creased frequency of heart action may be accompanied by a rise in blood pressure; the patient becomes unable to lie quietly in bed, suffers from frequent micturition and is very uncomfortable until finally worn out, he falls into troubled slumber. This disturbance of sleep is largely responsible for the peevishness of the dyspeptic. Longcontinued irritation of the abdominal sympathetics finds expression in mental and other nervous phenomena. The restricted dietary and consequent feebleness and anemia interfere with the usual physical activity and lead these patients to fear extremes of cold or heat. They are always seeking protection of one kind or another. muscles become flabby and elimination defective. some cases there is no loss of weight, and indeed there may be obesity, provided the intestinal digestion is not much impaired. On the other hand, a good deal of emaciation may occur, especially when there is considerable dilatation of the stomach. The character of the stomach contents of course varies in ratio with the digestive powers.

Some cases of chronic catarrhal gastritis are discovered to be such only through exclusion; a routine examination including that of the stomach contents does not suffice. These patients do not complain of the common

symptoms of the affection but seek medical advice with diverse complaints. Gastritis may exist without marked gastric symptoms. Large amounts of mucus are not necessarily present in the vomitus. There may be an excess of ropy mucus which originates in the esophagus and, although it is in no way related to gastritis, the patient believes that it comes from the stomach and that it is an index of a gastric condition. The mucus which arises from gastritis is intimately mixed with the food particles and may appear as shreds or flakes especially before marked failure of gastric secretion and digestion, but it does not occur in long, ropy strings such as are characteristic of esophageal mucus. There is an exception to this in cases in which the gastric parenchyma is transformed into mucus-bearing tissue. Often patients are deceived by nummular masses of mucopurulent material in the vomitus. Such masses have been swallowed and come originally from the upper respiratory tract.

Microscopic examination of mucus coming from the air passages will probably show the presence of pigmented alveolar epithelium that comes from the mouth and pharynx, the typical squamous protective epithelium, whereas the mucus of the stomach, in a case of gastritis shows the columnar epithelium belonging to the gastric When there is active digestion, that is, before the secretion of hydrochloric acid and ferments has materially fallen off, perfect epithelial cells are rarely found, vet the nuclei of these cells remain. On the other hand, when the digestive powers are insignificant, there are present cells unchanged by digestion. Although the microscopic evidences in catarrhal gastritis are scanty and often insufficient, one should not fail to appreciate what actually is present. The diagnosis depends so much upon results of the study of the contents of the stomach and

especially upon the character of the mucus that is found, that especial attention should be given to the microscopic The character of the gastric juice should examination. receive close attention. One should not be content with the results obtained from a single examination, but should study the secretion and the digestive power after repeated and varying test meals; also, there should be an inspection of the wash water obtained from the stomach during the hours of fasting. In a great majority of cases the hydrochloric acid, as well as the digestive power, will be decreased, but this varies from day to day. The intermittent, irregular but, on the whole, progressive decline in secretion is to be expected. A temporary improvement in the condition of the mucosa results in an improvement in the secretion, even though there is little change in the quantity of mucus. This rule, however important, does not invariably hold good, for the condition known as acid gastritis is occasionally met with. although in my experience much less frequently than is stated by some clinicians. I am convinced that cases of over-secretion which are temporary and functional in character are mistaken for acid gastritis. Admitting that gastritis may be temporarily associated with a secretion that is highly acid, we should expect to find the other features of the case to correspond with an inflamed mu-When cases of uncertain nature are encountered. a decision as to their real nature should be reserved while observing their behavior under treatment. When with hyperchlorhydria no improvement follows a proper regimen and other measures that would be expected to relieve a functional condition, when the gastric mucosa remains irritable and when there is uniformly present flaky or stringy mucus intimately mixed with the food particles or adhering thereto and when the nuclei of the epithelial

cells are discovered microscopically, then admittedly there is an acid gastritis. The secretion of pepsin and of rennin usually keeps step with that of hydrochloric acid, and when one is deficient the same may commonly be expected of the other. Sometimes, however, the ferments continue in almost normal proportion though the hydrochloric acid is very deficient. When we find the combined chlorids present out of proportion to the free hydrochloric acid, or when we find a good total acidity without free HCl, the acidity depending upon combined chlorids, we may assume that the ferments are present in sufficient amount. There is often a disagreeable odor from the stomach contents, the result of fermentation.

The tongue is large, soft, and shows the indentation of the teeth, and in no other gastric disease is the tongue so uniformly coated. The state of the bowels is inconstant. When the digestion is faulty and the ingesta reach the intestines in a condition too coarse and unprepared and irritating, there is caused not infrequently irregularity of the bowels and even diarrhea. Constipation, however, is often met with.

The blood and the urine show no changes that are characteristic of gastritis. There are only such variations as may be attributed to the nutritional and toxic disturbances.

So far as concerns the physical examination of the patient, there is ordinarily nothing to be discovered which is directly related to chronic gastritis, but this statement does not apply to stenosing gastritis or that rare disease which begins in the serous covering and results in thickening and contraction of the organ. Nevertheless, physical examination is important because of signs that may be discovered of atony, gastrectasis and motor insufficiency. These may have resulted from the gastritis and

they are, when present, so important that they should be searched for from time to time, not only as indications of the progress of the case, but because of modifications in treatment which would be entailed.

Almost unexceptionally in the course of chronic gastritis we find periods of improvement succeeded by periods in which all the symptoms are augmented. Generally we can account for these relapses by some indiscretion. dietetic or otherwise, on the part of the patient or by untimely discontinuance of the treatment. Relapses are frequently the result of intercurrent diseases and are often met with in connection with influenza. They also are the consequence of psychic or nervous disturbances, unwonted physical activity or neglect of the intestinal tract whereby constipation is induced with resulting hepatic congestion or auto-intoxication. In women the condition is often intensified at the menstrual period and with the nervous perturbation incident to the menopause. One should be alert in the course of chronic gastritis for the appearance of complications or for the onset of some other disease of the stomach. Of all intercurrent disturbances those which we most frequently have to deal with are the functional neuroses. Functional nervous troubles that create their own train of symptoms, which imitate very strikingly those of catarrhal gastritis, cannot always be distinguished from the manifestations produced by an inflamed and irritated stomach. It is always best to determine so far as possible how much of the complaint and which of the symptoms are referable to the nervous system. Of course we cannot always do this and errors are made even with exercise of the best judgment. The importance of this matter becomes evident when we hear patients recite a history of long suffering which has been erroneously attributed to neurasthenia or

eve-strain and when upon a study of the stomach contents we find there indubitable evidence of a chronic gastritis which has been overlooked. Another mistake which occurs still more frequently is shown in the story of those who have been undergoing interminable lavage, low diet and useless medication relief of a supposed gastritis, when study of the gastric contents assures us of a healthy mucosa. Diagnostic errors of this kind are not uncommon and one is on safe ground only when he adopts the routine practice of going over the case anew from time to time with an open mind and with a critical attitude. Also we must be watchful for signs of motor insufficiency which may announce an extension of the disease into the muscular tunic, with resulting atony and beginning dilatation; and we must be vigilant to recognize cancer which, coming on insidiously, may be the cause of delay at the pylorus. Also, occasionally, true peptic ulcer occurs in the course of chronic The fact that this is not more often the case seems to me strong ground for disbelief in the doctrine of an inflammatory and infective beginning of peptic ulcer.

Diagnosis and Differential Diagnosis.—The recognition of a chronic catarrhal gastritis must rest upon the presence of long-continued dyspeptic symptoms, markedly affected by the diet and the presence after test meals of gastric contents showing lowered secretion, imperfect digestion, mucus intimately mixed with the food particles, gastric epithelial cells, leucocytes, and an unusual abundance of bacteria. These symptoms associated with disturbed sleep, mental depression and irritability, with lowered nutrition and its attendant chain of symptoms, comprise the chief manifestations of chronic catarrhal gastritis. It may be distinguished from the dyspeptic symp-

toms depending upon purely functional derangement by the persistence of the symptoms and the sensitiveness which the stomach shows to any unusual tax put upon it. Although these symptoms may occur with a neurasthenic stomach, they rarely comprise such a definite group and do not follow such a characteristic course. The neurasthenic stomach shows vagaries with striking deviations in the quality of the secretion, unusual motor excitability or exaggerated sensory symptoms. It will be found that the gastric symptoms of neurasthenia are to a greater extent modified by influences that produce systemic nervous irritability or fatigue than is true with chronic gastritis. Perhaps the most important point of differentiation lies in the fact that in functional nervous disturbances the symptoms can be relieved with comparative promptness by suitable management; whereas when the symptoms depend upon an inflamed mucosa, they respond less readily to treatment so that it soon becomes evident that there is some local disturbance apart from. or in addition to, a perturbed nervous system. By recognizing symptomatic peculiarities and squaring them with the results obtained by laboratory examination of the stomach contents it is usually possible to find sufficient evidence for making a differential diagnosis. distinguish between early cancer and chronic gastritis may be difficult, particularly when the cancer develops in the course of chronic gastritis. The probability that cancer is present is strong when there is a persistence of symptoms in spite of measures of treatment that should relieve chronic gastritis. For instance when a suitable diet has been established or when lavage has been judiciously carried on for some days, simple catarrhal gastritis should be temporarily relieved; whereas when cancer is present little or no benefit will follow these measures. Unfortunately this statement is not invariably true, for the stomach symptoms in early carcinoma may temporarily disappear under this treatment. The improvement, however, is short-lived. We are usually able to obtain a clear conception of the situation because of a greater loss of weight in cancer and because of the unmistakable, although at first, almost indefinable cachexia. Previous to the occurrence of ulceration in malignant disease there is little to be learned in the search for occult blood in the stools or stomach contents. Sooner or later slight hemorrhage occurs and then the more delicate tests assist materially in reaching a diagnosis: especially is this true when blood is found in a case showing characteristic modifications in the gastric chemistry. When there is a progressive decrease in the hydrochloric acid, the early disappearance of rennin zymogen, the appearance of lactic acid and the Oppler-Boas bacilli in the stomach contents and the beginning of motor insufficiency, we have convincing evidence of cancer. There are many other facts of subsidiary importance concerning both in symptoms and gastric analysis. Among the symptoms may be mentioned an increasing capriciousness of appetite, distaste for meats and meat preparations and irregular behavior of the stomach in the matter of accepting certain foods, somewhat regardless of their digestibility. Among the gastric findings is an excess of albuminous material in the fasting stomach. Also there may occur albumose in the urine and deviations in the blood picture. In reviewing my cases of cancer of the stomach, I have found that 75 per cent were over 50 years old; there was absence of free hydrochloric acid in 75 per cent, its presence at least occasionally in 24 per cent and the presence of lactic acid in 60 per cent. Unfortunately for differentiation between cancer and gastritis, the cancer cases showed the presence of excess of mucus in over 80 per cent of cases and often the presence of leucocytes and red blood corpuscles, occasionally present in gastritis.

The differentiation between chronic gastric ulcer and chronic gastritis is usually less difficult. There are pain and localized tenderness; often increased secretion of hydrochloric acid and active digestion of proteids are found in the stomach contents of typical ulcer. appetite is generally unimpaired, unless there is motor insufficiency, and there is no distaste for meat. The greatest difficulty will be found in the case of pyloric ulcer causing moderate obstruction and stagnation. Under these circumstances an intercurrent gastritis is not an unusual event, and unless the stomach contents is examined often the nature of the case may be misconceived. Matters are yet more difficult when in connection with the motor insufficiency there is a decrease in gastric secretion, which condition we may encounter in exceptional cases of ulcer, even at the pylorus. Of importance in the differentiation between gastritis and ulcer is the continual finding in the latter of occult blood in the stools.

Between syphilis of the stomach and chronic gastritis we may decide by the use of the Wassermann reaction, by finding present the constitutional signs of syphilis and by the application of the therapeutic test.

Prognosis.—A recovery from chronic gastritis may be expected in uncomplicated cases of not too long standing and great improvement follows rational treatment, even when the gastritis occurs as a complication of really incurable affections. The disease is easily re-excited, and often a patient is discharged or gives up treatment before complete recovery, because relieved of symptoms. As a result of such recurrences the obstinacy of the affection

has come to be exaggerated. When gastritis is secondary to a persisting congestion of the stomach, a condition found often in connection with hepatic and thoracic diseases, it is difficult to cure and is disposed to relapse. There comes a time in the history of such cases when the venous stasis cannot be lessened, and the gastritis grows worse notwithstanding intelligent treatment, and frequently, because of resulting innutrition, is the direct cause of death. Most cases of chronic gastritis follow a somewhat irregular course. The symptoms become inconspicuous and the gastric contents may resume an almost normal condition; then the disease relapses again.

Many of these patients are subject to intercurrent acute gastro-intestinal attacks in which the striking symptoms are nausea, eructations of gas and regurgitation of stomach contents showing that certain articles of food have remained far too long in the stomach. Besides, there is a sensation of gastric distress, burning, weight, distension, tenderness or pain. There are borborygmi, perhaps griping intestinal pain and diarrhea; there are usually present evidences of acute intoxication. With a thorough clearing of the bowels with castor oil, followed by a few doses of subgallate of bismuth, or of bismuth salicylate and salol, and with abstinence succeeded by a careful and gradual return to the usual diet, all symptoms disappear. Indeed for a time the patient may feel unusually well and may continue to feel so for days or weeks according to his caution. Then there is a recurrence of the attack. I have known people to continue this experience Now, in fact, such patients are usually suffering from a chronic catarrhal gastritis; they are never free from it, even when feeling at their best; they are merely free from symptoms. Always the disease is present although latent, waiting to crop out when resentment is sufficiently invited. The attacks come on when the resisting power is depressed by some chronic infection or intoxication, such as sinusitis, prostatitis, pyelitis in the one instance, and uremia or lithemia in the other.

Treatment.—In undertaking the treatment of chronic catarrhal gastritis special effort should be made to ascertain the predisposing and underlying causes, and to remove them when possible. When it is not possible to remove the cause we may study it to advantage, seeking to divert the influences that make for gastritis. As illustration, let us instance gastritis developing in a patient with chronic pulmonary emphysema. Here we must expect great difficulty in any attempt to cure the patient but the symptoms can be controlled more promptly by requiring him to avoid such exercise or effort as would increase pulmonary congestion, to avoid inclement weather, to choose a suitable climate and to improve the general vigor by Swedish movements and hydrotherapy, hoping thereby to avoid bronchitis. Thus by eliminating the sources of pulmonary obstruction and hence gastric hyperemia the direct treatment of gastritis will prove vastly more successful.

Another example is the victim of uremia. Great attention must be given in his case that the intake of proteids is not beyond the metabolic and eliminative possibilities. He should be further assisted by a "chlorid low" diet and by procuring through vapor baths, friction, etc., a marked activity of cutaneous excretion. In case of hepatic insufficiency much may be gained by an occasional sojourn at Carlsbad or by the drinking periodically of mildly purgative water rich in magnesium and sodium sulphate. Cardiac insufficiency offers a most frequent illustration, as the accompanying gastritis will often improve under the use of digitalis or the adoption of a

course of medical gymnastics and hydrotherapy when measures directed immediately to the cure of gastritis may be unavailing. From this it will be understood that in any case of chronic gastritis we should not be content with treating the disease directly, but should employ all indirect methods which may be fruitful in removing underlying causes.

In the direct treatment of the disease that which is of greatest importance is a suitable diet: it is necessary to allow the full amount of food needed to maintain the balance of nutrition: therefore the number of calories ingested should be known to the physician and he should attempt to prevent a deficiency in any direction. patient with chronic gastritis, when he has a single unfortunate experience with a particular article of food usually eliminates it from his regimen and, perhaps, unwisely. By continuing this practice he may become gradually reduced to a diet so limited in quantity and in variety that unnecessary weakness results. Whenever a seemingly appropriate aliment disturbs a patient, it is wise to look carefully into the mode of its preparation, as therein is often found the source of disturbance rather than in the essential nature of the food. Whenever the gastritis becomes aggravated, that is, when there occurs an acute exacerbation, a complete fast should be insisted upon for a short time, followed when possible by a milk The milk should be fresh and pure; it should be given at intervals of two hours and is often best served hot, but not scalded. It should be sipped slowly, according to the excellent suggestion of Dr. Bulkley, never swallowed rapidly as one does a glass of water. The patient should be made to understand that milk is a food rather than a drink. Should milk prove disturbing, we may remove the cream, allowing merely skimmed milk. Its di-

gestion is sometimes enhanced by fermentation. purpose may be used the bacterial ferment of kephir, or the Bulgarian ferment advised by Metchnikoff. with lime water, milk slightly peptonized or shaken up well with a dilute solution of hydrochloric acid may agree well with the patient when ordinary milk is disturbing. Almost all thoroughly boiled gruels are well tolerated and may be substituted where milk disagrees. The cereals are to be given with discretion, for starchy food in this form, swallowed with imperfect insalivation, is often less well tolerated than when given in the form of stale bread. zwiebach, toast, etc. An effort should be made to enlarge the dietary, being governed as to the extent of the increase both in quantity and variety by its general effect on the patient as well as upon the digestion. Observation as to the state of digestion should be made by use of the stomach tube and by examination of gastric contents. The time required for the digestion of a given food, the quality of the gastric juice secreted and the rapidity with which the stomach empties itself should be known. following these precautions the dietary may be extended. thus increasing the resisting power.

With this in view it may prove desirable to review the ensuing list of food. Broths and soups, not too rich in fats, and purées may be given in almost all cases. The meat extracts present in broth stimulate gastric secretion and rarely cause distress. Beef and mutton are the meats oftenest tolerated but they must be properly prepared, for this makes a great difference; cooked fats should be avoided, therefore the meats should not be fried. The value of meats, long boiled and properly dressed seems not to be sufficiently appreciated. Next to boiled meats, those that are broiled or grilled stand high in digestibility.

In meats that are roasted or fried, the fats are so changed that peculiarly irritating fatty acids are developed. Only boiled ham should be allowed, and never pork or veal. Raw beef finely minced and taken as a sandwich agrees sometimes better than when cooked. Boiled fish only should be permitted and those having little fat selected. Yellow pike, black bass, perch, weak fish, sole or flounder are suitable, whereas salmon, mackerel, sturgeon, and eels are among those that should be interdicted. Fresh oysters on the shell are suited to most cases, not ovsters that are cooked. Very soft boiled eggs, or raw eggs stirred in a broth or gruel or taken raw with a few drops of lemon juice are usually well digested and offer an easy method of supplying a demand for proteids when there are objections to meat or fish. Mealy baked or boiled potatoes, served at once before they have had time to become heavy; skillfully prepared boiled rice, macaroni or spaghetti, and well-ovened stale bread, including that made from the whole wheat, constitute the most important of the starchy foods that should be used. There is no objection to hot bread provided it be stale; that is to say, it may be warmed over or toasted. Fresh green peas or beans may be added to the list and, in certain instances, selected cooked salad or greens, such as boiled lettuce, asparagus tips and very fresh young spinach. Care is required in the use of these green vegetables or so-called "fodder foods." Though sometimes well borne, they are again unaccountably unwholesome. Strange to say some patients take, without experiencing distress, uncooked tender lettuce, endive or celery, but as a rule these should be withheld. Fruits and fruit juices, especially when uncooked nearly always cause disturbance in gastritis. Coffee and cocoa should be interdicted, but tea is sometimes permissible. Wines, beers, and spirits must be ex-Water is the drink most suitable and some attention should be given to finding the spring that agrees best with the patient. Very often a good deal can be gained by taking water varying only slightly in its constituents from that of another spring formerly used; while one is suitable, another is not, and we may not see why. Waters containing the bicarbonates are preferable to those containing the carbonates. Slightly alkaline waters, like vichy, or those gently aperient, like some of the admirable Saratoga waters, are often useful when taken with moderation. Alkaline waters are suitable for cases with sufficient secretion or too acid contents, and weakly saline waters for those with deficient secretion. Generally speaking, water should not be taken during the repast, especially when there is low secretion and dilatation; exceptions to this occur when there is acid gastritis; then digestion is favored by drinking moderately with meals. The meal hour is a matter of consequence. At what hour should the most substantial meal be taken? pends upon circumstances. When duties permit, a sufficient period of rest should follow the chief meal of the day, usually best taken at noon; especially is this true when a full repast in the evening leads to insomnia or disturbed sleep. On the other hand, when the patient is necessarily active during the afternoon, it may be best to give the heartiest meal at six o'clock in the evening, exacting from him the practice of resting for a short time before and a long time after eating. When dinner is eaten at midday, the breakfast should be very light, and we must be satisfied that it is disposed of and the stomach empty and at rest before the hearty meal is taken. When, in order to introduce the proper number of food units, it becomes necessary to give a more bountiful

breakfast, it should be taken early in the morning. It is a good plan to have the patient take a glass of milk on rising, before dressing. An hour afterwards he should take one or two very soft eggs, a cup of tea and some fresh dry toast or bread with butter. During the forenoon, if hungry, he should be allowed a cup of hot broth with a cracker. With this arrangement the patient probably will be able to take his chief meal at midday without trouble.

LAVAGE.—Lavage is an important measure of treatment in chronic catarrhal gastritis, but it by no means effects a cure. Sometimes it should be practiced for several days in succession, and then omitted until the symptoms again demand it. In other cases it is best to employ lavage on alternate days or twice a week for an extended period of time. It may be most efficient when used at bed time, thus permitting the stomach to rest until morning. This is especially important in gastritis associated with motor insufficiency. Great benefit attends the use of massage, medical gymnastics and hydrotherapy in these cases. When the motor function is depressed, a few minutes' abdominal massage an hour after eating is very serviceable. More general treatment directed towards equalizing the circulation and stimulating the innervation should be employed daily or on alternate days when possible. As to what "agrees" with the stomach and what "disagrees," are not our opinions influenced too much by preconceptions? It would seem to be so. We carry with us the beliefs taught us when children, or acquired later in life through some notable experience which we have had or have heard of, or we have opinions resulting from what we find to be suited or unsuited to ourselves. The laity smile over our disagreement and with justice. There is no fundamental reason why one physician should be an ardent advocate of the milk diet and why another should treat it as quite unfit for an adult stomach. The same may be said of the meat diet so much insisted upon in the treatment of digestive derangement by certain well-known clinicians. The ill effect of fruit in poor digestion many of us have learned to fear, yet fruit is found to be admissible in some cases and others are benefited by the "grape cure." There is much exaggeration of truth and error in the matter of diet. It is of the utmost importance that we should be opportunists in prescribing the foods that our patients may be permitted, except where we are governed by unprejudiced observation and by physiological facts rather than fancies, by laboratory demonstration rather than routine habits of thought.

Drugs.—Drugs occupy a secondary place in the treatment yet at times are not only useful but necessary. For instance, fermentation can be corrected by small doses of the salicylates; distress can be overcome by the use of phenol, chloroform water or chloretone. Innervation can be improved by nux vomica and the simple bitters, and great advantage may attend the proper use of alkalies in some cases and mineral acids in others. When the stomach is unusually sensitive even a moderate gastric acidity occasions distress and then light calcined magnesia and bismuth are indicated.

When the gastric secretion is very low, local comfort and, doubtless, some assistance to digestion follow the taking of the mineral acids after eating. The best of these is the stronger hydrochloric acid, of which four or five drops may be taken with egg albumin in half a glass of water.

Phosphoric acid, as in the following prescription, suits some patients particularly well:

DRUGS 597

R			
•	Acidi phosphorici dil	8.00	(3ii )
	Acidi phosphatis	16.00	(3iv)
	Aquae destillataeq.s. ac	120.00	(3iv)

Sig.—Two teaspoonfuls diluted with half a glass of water half an hour after meals.

Some cases do not tolerate mineral acids even though there is a deficiency in secretion. There is unquestionably a place for the use of pepsin and other gastric ex-The pharmacologists have shown us that the digestive power of most preparations of the ferments is extremely feeble. Besides it is shown in the routine examination of the stomach contents, that the administration of pepsin with or following a meal, has a scarcely appreciable effect upon the digestion. Nevertheless, a proportion of patients seem to be made more comfortable by taking full doses of pure pepsin with or directly following the meal. I have no explanation to offer for this effect. In a few exceptional cases there was noticeable an improvement in the constitutional symptoms, which suggests that these substances may have some beneficial effect upon the economy of which we have no knowledge. Perhaps extract of the gastric mucosa may prove the most efficient of this class of preparations. It seems probable that we shall soon possess some means of introducing the hormones which increase respectively gastric secretion and motion. Framont and others have utilized therapeutically the gastric juice of living animals, obtained through a permanent fistulous opening into the stomach. I have had too little personal experience with this to speak authoritatively but I am convinced from the experience of others that it is efficacious. Granting the favorable reports concerning this, it is probable that its activity depends upon the presence of some other factor aside from the usual ferments and hydrochloric acid. The imperfect digestion of starch probably depends upon fermentation or defective insalivation or upon a deficiency of ptyalin in the saliva. It has been found that in gastritis there exists a correspondence between the decrease of the gastric secretion and that of the salivary glands. The fault may lie in the continued high gastric acidity which inhibits the action of the starch-digesting ferments. In that case improvement will follow the administration of alkalies with or immediately after the meals. For this purpose Aaron recommends the following.

Ŗ			
•	Sodii bicarbonatis	8.00	(3ii)
	Magnesii oxidi	8.00	(3ii)
	Calcii carbonatis		
			ÌМ́.

Sig.—Take one teaspoonful in a little water immediately after meals.

When the acidity is low there is apparently some benefit attending the use of pancreatic extract or of foods partly peptonized. Something appears to be gained by taking the infusion of malt or the malt extract. Although the actual physiological change observable in the stomach contents from the use of these preparations is small, yet with their administration patients find less discomfort from eating, are thus encouraged to take more food and hence obtain real benefit. When fermentation proves to be a disagreeable factor it may be controlled by irrigation of the stomach with a solution of salicylic acid 1-1000 or by magnesium permanganate 1-1000, or thymol 1-2000. Of these I regard salicylic acid as the most effectual. A more continued and on the whole more satisfactory effect, may be obtained by the administration of bismuth salicylate 0.30 (gr.v), once in four hours; to this may be added an equal amount of salol. The combination seems to be particularly efficacious when the intestine DRUGS 599

is also involved. The sulphocarbolate of zinc .12 (gr.ii) between meals is also useful.

Although better results may be obtained from the practice of lavage (for which should be used a normal saline solution or a solution of boric acid, 1-500,) than by the drinking of mineral waters, the experience of centuries would seem to warrant their use. Following the treatment by lavage, or when lavage is impracticable, "a course" at the springs may be advised. Among waters which have achieved an enviable reputation in this connection are those from the springs of Kissingen, Homburg and Wiesbaden, Carlsbad, Vichy, Chatel-Guyon, Saint-Nectair and Plombières. Several of the Saratoga waters and the water from the Alum Springs are also included in this list.

Other remedies are useful in the treatment of catarrhal gastritis, either owing to their bitter quality or because of some specific effect. Of importance in the long list of these are condurango, chiretta, eupatorium, wahoo and ipecacuanha (in small doses long continued). Other remedies are useful because of their astringent property; some of these are more efficient than others. Hydrastis and hamamelis are favorites and so are minute doses of zinc sulphate .015 gm. (gr.¼).

Arexin tannate .30 gm. (gr.v) has a stimulating effect on the appetite. Of all local remedies I have seen most benefit from ichthyol in a capsule, 5 drops given with equal parts of castor oil half an hour before meals and continued for a long time; in certain cases decided benefits result from small doses of iodoform given in a pill an hour before meals.

Bismuth subnitrate or subgallate, after lavage or several hours after meals are favorite remedies and justly so.

## ALCOHOLIC GASTRITIS

The action of alcohol in relation to acute gastritis is easily understood and already has received consideration. Chronic gastritis becomes in those who are addicted to alcohol a more complicated question. It is not always possible to separate the manifestations that are strictly local in origin from those that depend upon the constitutional effects of alcohol. The action of alcohol upon the gastric mucosa is that of an irritant, producing vasomotor dilatation; hence there may be induced prolonged congestion of the part. The appetite is deranged with the result that there is often distaste for simple and nutritious foods and a craving for those that are acid, stimulating or even irritating.

Through the dulling effect of alcohol upon the sensorium, there is a loss of appreciation of the more delicate shades of appetite as well as of abdominal sensations that are aroused ordinarily from irritation; hence the stomach is deprived of the guidance of the physiologic monitor and, as a result, it is frequently subjected to insult and injury. In the alcoholic there is quite universally present an hepatic hyperemia and inefficiency that contribute to already existing gastric congestion. Through the resulting imperfect activity in various other organs there is laid an unusual tax upon the function and structure of the stomach. For instance, cardiac excitement, respiratory deficiency, faulty elimination, and impaired metabolism contribute to produce an irritable or inflamed gastric mucosa.

Disturbance of the nervous system in this type of gastritis is constant and important, and because of this it is difficult to decide to what extent the symptoms come directly from the diseased stomach. For instance the vomit-

ing so prominent in the clinical picture is, to a certain extent, due to general or nervous rather than to local causes. When attempting to describe the symptoms of alcoholic gastritis we are immediately met by the difficulty of recognizing the purely gastric side of the picture. There are cases in which the general toxic symptoms are insignificant or, upon a casual examination, absent; yet the evidences of gastritis are very marked.

I have seen numerous patients who complained only of dyspepsia and whose general appearance was that of good health, yet there was present an alcoholic gastritis and immediate recovery followed the withdrawal of alcohol. It is important to recognize this fact, the more so for the reason that distressing stomach conditions may be induced in some patients by a small quantity of alcohol such as most people could imbibe with impunity. This susceptibility to alcohol on the part of a few is as remarkable as is the occasional immunity to intemperate drinking on the part of others. It is astonishing to observe no strikingly bad effects in the case of many "regular drinkers," who cannot be taken as criteria either of pathology or good manners.

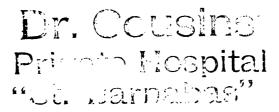
Symptoms.—In those cases in which the systemic effects of intoxication are scarcely noticeable, the dyspeptic symptoms probably depend upon gastritis, although functional disturbances are also to be considered. In some cases, apparently exempt from gastritis, prompt relief attends the withdrawal of alcohol. There is gastric irritability and almost always vomiting. So often does this occur on waking that the "morning vomiting of drunkards" has become a classical term. It may be the dominant symptom; the victim hastens to take his "drachm," which more often is twenty drachms, both as preventive and cure. There comes a day when the

specific fails: the drunkard is unable to retain the supposed infallible remedy, which stuff, in fact, is the cause of his disability. Unheeded, nature gives the same advice as the physician. Abstinence for a few days brings relief, but meanwhile the patient discovers new discomforts, gastric and otherwise. Anorexia, a sensation of burning, weight and sinking, nausea and eructations. water-brash, cardiospasm and hiccough become his symptoms. With the withdrawal of alcohol, insomnia, restlessness and peevishness enter upon the scene and tremor may be detected. More frequently we find gastritis but a part of the well-known symptom-complex of chronic alcoholism. The digestive symptoms may overshadow the others, or there may be so much general distress and psychic disturbance that less attention rests with the stomach.

Treatment.—In order to relieve the symptoms of alcoholic gastritis, it may be necessary merely to interdict the drinking. It is at times necessary to give the stomach a period of absolute rest, meantime putting the patient to bed and prescribing a sedative dose of chloral per rectum. It is remarkable how quickly the stomach will become quiescent and resume its usual powers upon withholding alcohol and inducing sleep. The rapidity of cure, except in dilapidated cases, leads one to suspect that the stomach symptoms are largely secondary and but a part of a systemic reaction or revulsion. However, examination of the gastric contents in old cases shows that there is an actual gastritis and there remain well-defined symptoms of dyspepsia even after the return of appetite. Therefore, we should insist upon a soft but highly nutritious diet in the fullest practicable amount, and should advise bismuth subgallate or the "gastric sedative," alkalies, strontium bromid and, when there are distressing symptoms, lavage. This last named procedure is not always carried out joyfully with inebriates, but it is nevertheless effectual.

In these cases we are not justified in confining our therapy to the stomach; the man requires attention and by treatment of a more general kind we indirectly relieve the gastritis. There are certain time-honored remedies which should be discarded; for instance, the permitting of small doses of alcohol, the tincture of valerian in doses of 15 c.c. (3 s.s.); the tincture of capsicum in water or in broth; a mixture of gentian, rhubarb and aromatic spirits of ammonia, with or without magnesium, and many These are more or less objectionable according to their action as local stimulants. Infusion of the simple bitters, strychnin and mild astringents have a place, and individual symptoms will demand special attention; but on the whole, the more simple the treatment the better the result, and the more easily may one mark the progressive steps towards recovery. The element of toxicity must be dealt with; therefore purgatives and diaphoretics should form a part of the early treatment almost without exception.

Finally, the treatment of alcoholic gastritis will usually prove commensurately successful with the treatment of alcoholism. Faithful abstinence from alcohol cures both the patient and his gastritis.



## CHAPTER XXVII

#### ACHYLIA GASTRICA

Nature and History.—The persistent absence of gastric secretion has come to receive the name of achylia gastrica, first given to it by Max Einhorn (1892).

The condition has been known since Flint (1860) described it as a result of atrophy, or degeneration, of the gastric mucosa and ascribed to it the cause of pernicious anemia. Many years later Fenwick 1 discussed the question under the title, "Atrophy of the Stomach," and pointed out its relations to anemia. In 1893 Allan Jones from my laboratory described the condition under the name of "gastric anacidity." The term achylia gastrica is descriptive and convenient and has the advantage of avoiding mooted questions of pathology.

It is known that in grave anemias, especially in the pernicious type, the gastric secretion fails and often disappears permanently. This occurs so frequently in pernicious anemia that Einhorn concludes that the relationship is special and that the disappearance of gastric juice in pernicious anemia should not be grouped with cases having another etiology; yet I see no necessary reason for placing these cases in a class by themselves.

In 1904 2 I reported a study of pernicious anemia and its relations to gastric digestion, based on 25 cases. Of these cases 22 showed absence of free hydrochloric acid and 3 of combined chlorids. It will be seen that achylia

<sup>&</sup>lt;sup>1</sup> Lancet, July 7, 1877.

<sup>&</sup>lt;sup>2</sup> Jour. Med. Assoc., July 16, 1904.

gastrica is present in about 80 per cent of the cases of pernicious anemia. On the other hand there is found a preponderance of cases of achylia gastrica which are unassociated with anemia or, if connected, merely with that of a moderate secondary type. It may be concluded that the atrophy of the gastric mucosa which occurs in pernicious anemia is not the cause, but rather a result of that remarkable disease. The activity of the salivary ferments is also impaired as is shown in the fact that starch digestion is poor.

A summary of the report before alluded to is here introduced:

Of the 25 cases of pernicious anemia reported, the youngest was 32 years old, the oldest 70; average, 45 years, there were 20 males and 4 females; 13 had outdoor occupation and 11 indoor; the longest duration of illness was in one 5 years, the shortest 6 months and fatal; 12 are known to be dead, 5 living, the others unknown; all had dyspnea on exercise; all suffered from faintness and weakness; 16 had gaseous pulse, 8 did not; 16 had heart slightly enlarged, 8 did not; 16 had systolic apical bruit, in 8 it was absent; 6 had bruit de diable, in 18 it was absent; 8 had other bruit; 16 had no other; 22 had a distinct lemon-colored tint of the skin, in 2 it was absent; 15 had the tongue strikingly denuded of epithelium, 9 did not; 10 had good appetite, 7 capricious, and 7 anorexia; 18 complained of symptoms of stomach trouble, 6 did not: 10 had constipation, 8 diarrhea, and 6 irregularity of the bowels; in 10 the liver was enlarged, in 13 it was not enlarged, small in none; 1 had four periods of improvement; 3, two periods; 8, one period, and 8 no period of improvement. After my attention was called to the subject by Billings' paper, I found signs of spinal cord involvement in 6 out of 9 cases. Nine cases showed slight dilatation

of the stomach; in 13 the gastric digestion was absent, in 8 it was very low, in the 3 others it was fairly good. showed evidences of gastric catarrh; in the others it was absent. Six showed no acidity of the gastric contents, 1 (the highest) showed a total acidity of 46, one 26, and the average about 10. Four showed the presence of combined chlorides: in the others it was absent. One showed free hydrochloric acid, .05 per cent, one .06 per cent, and one .022 per cent; in all the others it was absent. showed lactic acid present; in the others it was absent. Four showed other organic acids; the others, none. acid salts were very low, 18 being the highest, 5 having none whatever; 7 showed biuret reaction; six showed rennet present, in the others it was absent. The digestion of starch was poor in all save two cases.

The absence of gastric secretion in pernicious anemia was attributed early to degeneration of the glandular parenchyma, perhaps from gastritis, perhaps from other causes.

With the practice of systematic investigation of the stomach contents, it was found that the gastric secretion was greatly modified by what appeared to be merely functional derangements. For instance, it was ascertained that in certain cases there occurred oscillations in the secretion, between a high and a very low standard.

Some patients were found to have over-secretion for a long time and subsequently to have low secretion or even temporary absence of all secretion. In these cases no evidence of gastritis or other structural changes were forthcoming. Under these circumstances achylia was, and still generally is, attributed to functional perturbation. The question arose, may not achylia gastrica in certain cases be merely the expression of a long-continued functional depression? Even in cases that showed at

necropsy a destruction of glandular parenchyma, it was believed that this might represent long ensuing functional inactivity, an atrophy from lack of use corresponding to the ocular atrophy in a disused eye.

Martius would divide achylia gastrica into classes: (1) those that are secondary to definite inflammation or degeneration of the mucosa, such as occurs in gastritis, carcinoma, etc.; (2) those that depend upon a primary. perhaps congenital, defect in the secreting structures. The mucosa in these latter cases he regards as especially susceptible to untoward influences and prone to gastritis. Einhorn 3 says: "The secretory functional disturbances are not based on a primary change in the mucous membrane of the stomach. They rather produce, if they last for a long time, lesions of the mucosa of greater or less extent." Riegel asserts: "It cannot be denied that occasionally achylia gastrica may be merely a perversion of function." Recently, Faber 4 has expressed the opinion that continued disappearance of secretion is based upon degeneration of the gastric mucous membrane, a view long since held by Ewald.

In certain cases the clinical course lends strong support to this view of structural change. Yet, I have studied and reported cases that continued for a year and were ultimately restored to health. This does not prove that these patients did not have a low grade of gastritis, although there was no evidence of it. While I have leaned towards the functional explanation of one group of cases a recent critical analysis of all my cases makes me feel that eventually the mucosa undergoes structural changes, but I do not conclude that an identical pathological march occurs in all cases.

<sup>&</sup>lt;sup>8</sup> Am. Jour. Med. Sci., Oct., 1902.

<sup>&</sup>lt;sup>4</sup> Zeit. f. Klin. Med., 1908.

We admit that there are definite periodic derangements of the secretion of the stomach that appear to be purely functional. Büttner<sup>5</sup> for instance, recognizes among others a periodic hypersecretion without perceptible basis in morbid anatomy. I have found a corresponding temporary disappearance of the gastric secretion to follow the same course and under apparently the same conditions. Why not a more persistent state? Is there any reason why there should not be, through physiologic disturbance, a permanent absence of secretion as well as a permanent hyper-secretion?

It has been shown experimentally that quite apart from the action of the nervous system, the secretion of gastric juice is excited through the action of a hormone developed in the walls of the stomach, passing thence through the circulation, and ultimately activating the secreting glands of the gastric mucosa. Hemmeter announces that a hormone having a like action is derived as an internal secretion from the faucial tonsils. It is not improbable that a deficiency in the gastric hormones is involved in achylia gastrica.

It may be concluded that achylia gastrica is not a disease per se; it is rather a loss of gastric function that may result from degeneration of the mucosa or from yet more obscure reasons. However, we must remember that the loss of function may eventually entail structural changes.

Etiology.—It is of most impotrance to consider those conditions that contribute to the development of achylia gastrica. For that reason I have carefully reviewed my private cases recorded during the past fifteen years and here present the results. Some of these cases do not fall under the strict definition given of achylia gastrica.

<sup>&</sup>lt;sup>5</sup> Arch. f. Verd. Krank., June, 1909.

That is to say, I have included cases in which traces of secretion occasionally were found. The reason for this is that these cases appear to represent the condition in the formative stage; in other words, where the secretion of hydrochloric acid and ferments is practically abolished and where their permanent disappearance may be confidently expected. It is impossible to include in this review all the opinions that have been reached on this interesting subject.

It will be seen that all agree that when the parenchyma of the stomach has undergone sufficient degenerative change the secretion disappears. As to whether this persisting loss of secretion may depend upon a functional depression alone, not preceded by structural disease of the stomach, is yet a controverted point, but the weight of evidence is in the affirmative.

A Phase of Achylia Gastrica.—There would seem to be some relationship existing between achylia gastrica and erosion of the gastric mucosa. In the latter condition there is usually subacidity, although this is not invariably the case. Sometimes there is a complete absence of gastric juice, or at best only traces of combined chlorids and evidences of slight secretion of the enzymes. It has been suggested by Dr. James Taft Pilcher 6 that these cases of gastric erosion depend upon the lowering of gastric secretion. He records a number of cases studied at Mayo's clinic in which he found the absence of HCl together with the presence of occult blood in the stomach contents. He looks upon these cases as distinct from achylia gastrica, but the reasons offered are scarcely convincing. He would explain the condition on the grounds of a reflex inhibition of the gastric secretion, usually secondary to some local exciting cause in the ab-

<sup>&</sup>lt;sup>6</sup> Jour. Am. Med. Assoc., Nov. 19, 1910.

domen, particularly appendicitis, cholecystitis and pancreatitis. The next step is the invasion of the stomach by pathogenic microörganisms the entrance of which is made easy through the disappearance of the normal gastric juice. The infection of the stomach, Dr. Pilcher believes, explains the erosion and the occult hemorrhage. He reports a remarkable experience in which these cases have recovered as a result of surgical intervention whereby there has been removed the supposed exciting cause, that is to say, appendicitis, cholecystitis, etc.

According to my experience it is common to find in cases of erosion of the stomach a low gastric acidity or achylia sometimes with occult blood present together with leucocytes. There is more than the ordinary display of bacteria both in number and variety, but these cases have not been, so far as I have been able to follow their history, more frequently related to extra gastric abdominal lesions than is true of other affections of the stomach, nor have I found that cases of this character are numerous. Achylia gastrica without erosion, large numbers of bacteria and occult blood is far more common, and I cannot admit that the invasion of the stomach by bacteria depends upon the absence of HCl, for in the large number of cases of achylia gastrica which I have seen, the number which gave evidence of infection of the stomach was comparatively small. In fact, one of the striking features of achylia gastrica is that the stomach shows little defect save that which relates to its functional secretory activity. It is true that the condition of gastric erosion is often related to infection. I am not certain that the infection initiates the erosion. In cases of gastric erosion the mucosa of the stomach appears to be unusually friable. Particles of mucosa are easily dislodged by the end of the stomach tube and following this there is bleeding. That this can be produced by an infection of the stomach is not difficult to understand, especially when bits of mucosa recovered in the wash water show evidence of inflammatory infiltration. It is possible that the mucosa in this condition is more susceptible to infection than is true of normal tissues and the finding of unusual number of leucocytes and bacteria in some cases together with occult blood makes this probable. I have seen cases of erosion of the gastric mucosa appearing in patients who suffered from pyorrhea alveolaris. Here the constant swallowing of pus and pus-making bacteria may have accounted for the finding of these in the stomach contents, and that the mucous lining of the stomach would be invaded by these microörganisms is not improbable. There are other sources from which the stomach might easily become infected as for instance disease of the nasopharynx and dental caries. On the whole something is to be said for the doctrine that an unhealthy gastric mucosa may become invaded by pathogenic bacteria, yet I am not disposed to attribute this invasion by bacteria to the primary disappearance of the gastric juice, but rather to an antecedent unhealthy condition of the lining of the stomach dependent upon causes to me unknown, and to a loss in immunity to certain bacteria. Briefly stated gastric erosion may occur with or without achylia. It is often accompanied with slight bleeding; it is sometimes associated with direct infection of the digestive tract above the stomach. I am satisfied that it is not necessarily related to definite infection in the abdominal. cavity.

In the exhaustive study of the subject by Faber and Lange,<sup>7</sup> they examined cases such as I have considered in which gastric juice had practically lost its digestive

<sup>&</sup>lt;sup>7</sup> Zeit. f. Klin. Med., 1908.

power but where occasionally there was the presence of traces of acid and of the ferments. They found that the motor function of the stomach was usually increased, but occasionally deficient, and 50 per cent of the patients were free of symptoms, while in the remainder there occurred cardialgia, nausea, vomiting, etc. In only 2 of the 12 cases were there normal movements of the bowels. the others had constipation or diarrhea, usually the latter. The anatomical study of these cases showed that in some there was chronic gastritis, atrophy of the gastric mucosa, sometimes slight, sometimes far advanced; but in others the gastric glands were normal in appear-There was found one case of achylia occurring in pernicious anemia in which coincident with improvement in the blood, there was a return of gastric secretion. This is contrary to my own experience in the study of the stomach in at least 50 cases of pernicious anemia. These investigators conclude that achylia gastrica may exist as a purely functional disease without visible anatomical foundation, but that it also accompanies inflammatory and degenerative diseases of the gastric They agree with my conclusions that achylia is mucosa. especially a disease of later life and that clinically the functional type of the disease can not be distinguished from achylia due to atrophy or other diseases of the mucosa.

Treatment.—A positive but limited benefit in cases of insufficient gastric secretion is obtained by the introduction of the gastric ferments and hydrochloric acid. It is universally recognized that though these substances are moderately useful, they are lacking in efficiency and by their use alone the functional power in gastric digestion cannot be restored. It has been a matter of speculation as to why these substances in cases of limited secretion

are not more efficient than they are. In this connection it is interesting to recall the results obtained by Tramont by the use of the gastric juice obtained from the stomachs of dogs. There can be no doubt of the favorable results which followed the use of this substance and its action has been verified by others. planation of the benefit which was observed after the ingestion of the animal gastric juice compared with that after taking solutions of pepsin and hydrochloric acid led to the suspicion that there was secreted by the stomach some other substance or substances in addition to the known ferments and hydrochloric acid. In support of this idea is the discovery by Edkins 8 of a specific excitosecretory substance which is formed in the walls of the stomach, carried thence through the general circulation and back again to the gastric mucosa wherein it excites active secretion of gastric juice. Edkins secured this substance by boiling the pyloric mucosa in acidulated When this preparation was injected into the jugular vein without having been carried first through the arterial circulation it was capable of producing a very active flow of gastric juice without the introduction of any other agent in the stomach.

In our pharmaceutical armamentarium we lack this specific "gastric stimuline," unless we consider it available in the gastric juice of dogs, and assume the efficiency of this to depend upon the presence of "gastric stimuline."

Recently, Maurice Hepp of Paris has described his experience in securing the pure gastric juice from a pig. The secretion as secured by him is uncontaminated with food. Through a clever operation which results in the comparative isolation of the stomach, he has devised a

<sup>&</sup>lt;sup>8</sup> Jour. of Phys., xxxiv, 1906. 
<sup>9</sup> Am. Med., Oct., 1910.

method of securing the gastric juice without interfering with the general health and nutrition of the animal. Hepp announces that he has had very satisfactory results from the use of this substance, and that it is capable of restoring even long depressed gastric secretion. Whether or not the reputation of this remedy will grow with the trial now being given, its results thus far are instructive.

### CHAPTER XXVIII

# ARTERIOSCLEROSIS—INVOLVING THE VESSELS OF THE STOMACH

The symptomatology produced by sclerosis of the abdominal vessels is varied and important. Disturbances that are supposed to be functional in character, as well as definite structural diseases are often actually based upon arterial disease. The subject has been investigated sufficiently to show the relationship that exists between arteriosclerosis and clinical manifestations, but owing to the fact that demonstration has often to wait upon post-mortem findings, the precise incidence of abdominal arteriosclerosis is a matter of conjecture. Some years ago Teissier of Lyons called attention to aortitis and peri-aortitis below the diaphragm and showed that the sclerotic changes involve not alone the aorta but its branches and are accountable for degenerative changes and functional depression in the kidneys, liver, intestines and stomach.

The histological studies of Gestreich of Berlin show that of the vessels of the stomach the coronaries are most frequently and most extensively involved, and the large branches more frequently than the arterioles of the submucosa. In only exceptional cases he found the smaller branches to be sclerotic, while the larger vessels escaped. On the other hand, Grossman <sup>1</sup> found vascular and perivascular changes in the smallest vessels which are supplied by the sympathetic nerve fibers. He believes that

<sup>&</sup>lt;sup>1</sup> Arch. f. Verd. Krank., Aug., 1908.

the pain experienced in these cases is thus explained. It is noticeable that a single arterial branch may show advanced sclerotic changes although other vessels are free.

For this reason a clinical diagnosis becomes very difficult. Minute aneurysms may develop and, rupturing, give rise to profuse hematemesis or bleeding per rectum. Such a case is reported by Gallard and Buday, and a remarkable instance shown by H. U. Williams of Buffalo is referred to in the article on Gastric Ulcer. Even without gross pathological changes but with widespread. though inconspicuous degeneration, the vessels seem to have an over-sensitiveness to vasomotor impulses, resulting in periodical spasm associated with high blood pressure, a condition which has been especially studied by Pel. We too often forget that vascular disease may be at the root of intractable gastric symptoms whose etiology has thus far eluded us. Among the symptoms which have been found to depend upon arterial disease, pain is prominent. It may depend upon spasm or upon persistent narrowing of the vessels, the result of advanced sclerosis. In the latter case, the gastralgia develops two or three hours after eating and is especially prone to occur after physical exertion. At other times the attacks resemble those of angina pectoris save that the pain is in the epigastrium. Such attacks not infrequently are without assignable cause, occur at irregular intervals and very often at night. They resemble the gastric crises of tabes; they may continue for a few minutes or for hours.

Cases illustrating these symptoms of gastric arteriosclerosis were reported by me in 1903.<sup>2</sup>

Case No. XXVIII. Arteriosclerosis, Pain After Eat-

<sup>&</sup>lt;sup>2</sup> "Arteriosclerosis and the Digestive System," Albany Med. Ann., March, 1903.

ing.—An aged physician suffered from precordial and abdominal pain and from dyspneic attacks after eating. He was relieved by vomiting. He learned that the taking of starchy food or the eating too heartily of solid food brought on the attacks. He improved under frequent doses of nitroglycerin and the regular use of the iodids. Subsequently he informed me that he was able to eat provided he did not exercise, and to exercise provided he did not eat. In this case there was probably involvement of the coronary vessels of the heart as well as the abdominal vessels. In some instances it is difficult to determine whether the trouble arises in the heart or in the branches of the abdominal aorta. In several cases of rupture of the left ventricle secondary to arteriosclerosis producing focal myocarditis, I have found attacks of intense epigastric pain, and in some of these cases surprisingly little disturbance of the action of the heart itself. Of course, these cases ended speedily. The diagnosis of cardiac trouble was verified at autopsy, yet the patient's complaint having been primarily of pain, epigastric rather than precordial, the trouble might have been wrongly attributed to disease of the abdominal vessels. The pain of arterial disease is usually located over the celiac plexus or in the region of the umbilicus. This is illustrated in the following case.

Case No. XXIX. Middle-aged Man, High Blood Pressure, Gastralgia, Probably Syphilitic Arteritis.—A middle-aged man had vascular over-tonicity but an absence of the usual signs of arterial sclerosis. Syphilis was denied, but there had probably been a spirochete infection. He had suffered from abdominal pain accompanied by distension, often coming on at night. The stomach contents showed a total acidity of 111 with free HCl, 71, or 25 per cent. He was not relieved by the usual treatment for

hyperchlorhydria, but was distinctly relieved by nitroglycerin. This patient for whom morphin hypodermically had been necessary for the relief of nocturnal pain was cured of his trouble by the use of nitroglycerin and iodid of potassium; seven years later he reported himself well.

Another symptom of abdominal sclerosis is tenderness of the abdomen with a decrease in peristalsis and with distension. This distension contrasts with the retracted abdomen usually present in the abdominal pain due to lead poisoning and tabes.

Diagnosis.—The chief clinical manifestations of abdominal arteriosclerosis are pain, often paroxysmal and intense, at the epigastrium or at the umbilicus; distension of the abdomen accompanied by tenderness to pressure, diminished peristalsis, obstinate constipation, sometimes high blood pressure, occasionally severe hematemesis or melena.

Treatment.—Nitroglycerin and the other nitrites usually relieve the abdominal pain, although the benefit is sometimes of short duration. Too early resort to morphin should be resisted. Kaufmann and others recommend the use of theobromin or theobromin sodium salicylate, which is reputed to relieve the pain and other symp-Hamburger of Chicago has shown that while small doses of these preparations increase the arterial tonus, there is a distinct lowering of blood pressure when theobromin and its congeners are employed in full doses. There can be no question as to their efficiency in relieving symptoms. Where the arteriosclerosis is not too far advanced and when there is ground for suspecting an ancient syphilitic infection, mercury should be used, but sometimes more immediate, if less permanent, benefit is obtained through the liberal use of potassium iodid.

patient should be enjoined to take small meals of easily digested food and to refrain from exercise at meal hours. Great benefit follows a course of hot bathing and the application of hot abdominal packs.

## CHAPTER XXIX

#### SYPHILIS OF THE STOMACH

Ewald evidently regards syphilis as having little place in the etiology of gastric ulcer; Paul Cohnheim, on the other hand, says that in men it is an important etiologic factor. Dieulafov also believes it safe to assume that when the two diseases co-exist the ulcer is dependent on My own experience accords with that of Ewald. I have never been able positively to attribute a true peptic ulcer to the effect of syphilis, although I have found the affections concurrent. This simultaneous occurrence is but natural, considering the frequency of the two diseases. I have known peptic ulcer in the luetic to improve under specific treatment, but this is no proof of etiologic relationship between ulcer and syphilis. Doubtless the improvement in the general health assists in curing the ulcer. We do occasionally meet with syphilitic ulceration of the stomach, though rarely. Most often this results from a broken down gumma. Flexner cites a case where ulcer of the stomach followed a softening submucous gumma with obliterating endarteritis in the surrounding vessels. This hardly warrants his expressed belief that the relationship is frequent. There can be no doubt that syphilis may involve the stomach directly and that indirectly it affects it through disturbance of the general health. Fenwick names three modes of attack; viz., by the formation of gummata, by endarteritis and by chronic inflammation of the mucosa. Einhorn also admits three modes of expression, viz: ulcer, tumor and

stenosis of the pylorus. Most modern observers refer to gastric syphilis as a rather unusual event. In 1894 I reported 12 cases of unsuspected gastric syphilis which occurred among five hundred consecutive patients suffering from stomach diseases. For the most part these 12 patients complained of sensory and motor disturbances, regurgitation of gas, nausea, vomiting, distress after eating, delayed motion and especially gastralgia. In the succeeding fifteen years, I have added greatly to the number of these cases and have reached the conclusion that the symptoms, with the exception of gastralgia, are to be explained best by referring them to syphilitic lesions involving the gastric mucosa. Paroxysmal gastralgia in the majority of cases is to be regarded as a gastric crisis secondary to syphilis involving the posterior column of the spinal cord; vet gastralgia of minor importance apparently depends upon lesions in the stomach and may be attributed to ulceration, gummata or obliterating endarteritis. The frequency with which gastralgia develops in the syphilitic is a matter of special note. A series of such cases has been reported by Allan Jones.

Syphilis of the stomach may give rise to palpable tumor from hyperplasia, the formation of a gumma or a mass of adhesions involving the peritoneum, and may be accompanied by pain, hemorrhage and vomiting. It will be seen that the possibility of syphilis must be considered in the differential diagnosis of cancer of the stomach, peptic ulcer, chronic gastritis and the gastric neuroses. In differential diagnosis it may not be easy to exclude syphilis without resorting to the therapeutic test. When I find a patient suffering from persistent visceral disease the source of which is doubtful, who at the same time presents evidence of syphilis even though ancient,

I subject him to a thorough course of mercury by inunctions or hypodermic injections, and the transient use of potassium iodid by the stomach. To this is occasionally added a purgative dose of calomel. If under this treatment the patient does not improve rapidly, gastric syphilis is excluded as the cause of the symptoms.

The following history illustrates the type in which the symptoms are exclusively digestive, yet are not relieved by the methods of treatment ordinarily successful.

Case No. XXX. History of Syphilis Early in Life, Dyspepsia and Lowered Digestive Power, Cured by Injections of Mercury.—C. W. G. had a suspicious lesion fifteen years ago, was treated for syphilis and believed himself cured; he complained of gastric distress, eructations of gas and intestinal indigestion, and although the appetite was good he was losing weight. One and a half hours after an Ewald test breakfast, 300 c.c. of stomach contents were recovered by aspiration; total acidity of 32; HCl, 14; combined chlorids, 16; acid salts, 2. There was moderate atony but no evidence of gastritis. Gross inspection of the stomach contents showed poor digestion. and examination of the stools showed faulty intestinal digestion. He was treated by hypodermic injections of mercury. Four months later the gastric secretion had improved, he had gained nine pounds in weight and was exempt from symptoms. In some cases the treatment must be long continued before the stomach trouble is controlled, even though improvement is evident. other words, the stomach will continue to suffer symptomatically from the more general and constitutional ef-In these cases apparently the fect of the disease. symptoms do not depend upon a local lesion in the stomach, and may arise through disturbance of the nervous system, including mental worry, and in that sense may

be regarded as neurotic or psychopathic. This group of cases is familiar to all; they are regarded as "syphilophobiacs" and with good reason; however, there is often uncured syphilis in the background. Not infrequently syphilis is only one of several factors producing the symptoms, and besides worry we should take into consideration the possible excessive use of alcohol, the presence of anemia, and incompetence of the liver and kidneys.

There is a large group of cases in which the symptoms are gastric, but in which the liver is found to be large or irregular in contour. The deformity may be so slight as to escape any save the most painstaking physical examination. Then there naturally arises the question of malignancy for which syphilis is often mistaken. In this connection it would seem valuable to report the following cases from the wards of the Buffalo General Hospital.

Case No. XXXI. Gastric Pain. Slight Abdominal Induration, Normal Stomach, Negative Wassermann, Exploration, Cancer Suspected, Later Positive Wassermann, Cure by Mercury.—A lumberman, aged 45, single, American, complained of pain in the region of the pylorus, extending thence to the right costal margin, and believed that the trouble arose in his stomach. Nothing pathologic was revealed upon careful physical examination. man denied previous illness and gave no history of syphilis. The Wassermann reaction was negative. He had a leucocyte count of 8,200, hemoglobin of 90 per cent, and a normal differential count. Repeated examinations of the stomach contents and stools showed good digestion; an X-ray examination was negative; a period of rest in bed gave no relief. A course of hydrotherapy was tried, and various drugs were used without benefit. Reluctantly, a surgical exploration was requested, on suspi-

cion of an irritated gall-bladder. Upon section by Dr. Park it was found that the appendix and gall-bladder were both normal. Scattered through the liver were several masses which at the time were regarded as carcinomatous. Stomach and duodenum were perfect. Upon recovery from the operation the man was discharged as beyond help. However, his state of health did not materially change, and nine months subsequently at Dr. Park's request, he returned to the hospital. was still complaining of the pain but there was no advance in the supposed cancer. The slow course of the disease seemed now to exclude cancer, whereupon another Wassermann was made, which, this time, was strongly positive. The man was given mercury and he is now improving. Here is a case wherein the chief symptom was gastric pain, where syphilis was suspected but excluded along every line of investigation excepting the therapeutic test.

Case No. XXXII.—A Hungarian tinsmith, aged 48, had typhoid when 23; no subsequent disease. He categorically denied venereal disease. He complained of shortness of breath and dyspnea, but especially of anorexia, nausea, vomiting, sour stomach and gastralgia from which he suffered continually. The hepatic dullness measured seven inches in the mammary line; the contour of the liver was slightly irregular; there was cachexia and the abdomen was distended. The blood examination showed 30 per cent hemoglobin, a low red count and leucopenia. The stomach contents and stools on re-. peated examinations showed good digestion; the urine was highly colored and somewhat low in urea and total solids. The Wassermann reaction was negative and the classical signs of syphilis were absent. The diagnosis of several expert observers was carcinoma of the liver.

Before discharging the patient, in the light of past experience, it seemed wise to subject him to specific treatment. During the first fortnight there was little change, but thereafter the patient steadily improved. He gained 24 pounds in weight, the liver returned nearly to normal size, the blood improved greatly and the dyspeptic symptoms entirely disappeared. After a thorough course of mercury by the hypodermic method, the man was discharged practically cured.

These cases demonstrate how easily syphilis affecting the stomach and liver may be mistaken for malignant disease. In one, although the examinations were elaborate, nothing conclusive was learned until after the surgical exploration, the results of which were misleading. In the other, the indications of carcinoma were striking while the history, the negative Wassermann and the absence of characteristic signs seemed to exclude syphilis; yet the therapeutic test proved the diagnosis and cured the patient.

While nearly every description of digestive symptoms may be with propriety ascribed to syphilis, it seems advisable to emphasize pain. Although far from invariable, it is notably frequent and is often attributed to cancer, peptic ulcer, cholecystitis, appendicitis, etc. The character of the suffering is variable, and therefore deceptive and ranges from a dull ache, such as might accompany gall-stone or floating kidney to terrific and paroxysmal gastralgia, generally taking the form of the so-called "gastric crisis" which deserves further consideration.

Gastric Crises.—In 1866 Delamarre drew attention to "Des Troubles Gastriques dans l'Ataxie Locomotrice," which since Charcot's classical description have been termed "gastric crises." These attacks when typical

are characterized by a sudden onset of intense abdominal pain which often radiates upwards affecting the region of the sternum, sometimes the back and shoulders, and which at times is accompanied, or preceded, by the familiar lancinating pains in the lower extremities, or by the "girdle pain" characteristic of early tabes. dition, there is a sensation of gastric unrest and flatulence, ending in uncontrollable vomiting. At times, the vomitus is composed of highly acid juice, but toward the end of the attack, the acidity falls. The vomiting is often difficult and accompanied by retching. In amount it is sometimes very large, at other times small. rary relief of pain follows each act of vomiting. cessive salivation is seen as well as sweating, prostration and nervous excitement. Patients toss continually, the character of the pain being especially unbearable. The pulse is much accelerated, rarely retarded, and there is no fever. These seizures may continue only for an hour or two, but sometimes persist for days and even weeks, disappearing as suddenly as they began. They recur sometimes after a few days, again not for months or years. When these paroxysms of suffering are prolonged and oft repeated, the patient falls into a pitiable state of weakness and nervous depression. which in part depends upon the lack of nourishment but chiefly upon the pain. In rare instances the seizures terminate in a profound syncope with convulsions during which the patient sinks into an alarming condition. Often the attacks are less severe and closely resemble the painful syndrome met with in gastroxynsis (Rossbach), or gastrosuccorrhea periodica, the gastric symptoms of arterial sclerosis and other types or periodical vomiting. (See Cyclic Vomiting, page 643.)

Gastric crises occur in early tabes and may be the first

manifestation of the disease. A critical examination, almost invariably reveals other evidences of posterior sclerosis. In several cases I have noted exaggerated patellar reflex, although sometimes the pain is delayed until the deep reflexes have disappeared. In a goodly proportion of cases no history of syphilis was obtained, although proof of its existence was not lacking. Eventually after five or ten years the gastric crises fail to return; the later attacks may be milder than the early ones, with longer intervals of relief. This condition has been admirably described by Seymour Basch,<sup>1</sup>

Treatment.—Occasionally the pain may be relieved by large doses of antipyrin, or aspirin, by santonin combined with phenalgin, by "enesol," by frequent doses of chromium sulphate .30 gm. (gr.v) q. 2 h., or by salvarsan, but often like other tabetic symptoms, it is intractable to treatment and can only be relieved by morphin hypodermically which treatment is a frequent source of morphin addiction. In a considerable group of cases followed for years, while the gastric crises have gradually diminished in severity and frequency, the morphin habit has persisted. Occasionally it is possible to check the disease by the timely use of salvarsan, salvarsanized serum via the spinal canal, mercury and iodin.

<sup>&</sup>lt;sup>1</sup> Med. Rec., Oct. 14, 1899.

## CHAPTER XXX

#### PYLORIC STENOSIS OF INFANCY

The hypertrophic stenosis of the pylorus occurring in infancy is an affection characterized anatomically by hyperplasia of the muscular wall of the pylorus; functionally, by the more or less marked impermeability of the pyloric canal, and clinically by the development of uncontrollable vomiting accompanied by food stagnation, exaggerated peristalsis, constipation, diminished urinary secretion and progressive emaciation. The nature of this trouble is still a debatable question in pathology. Apparently more than one condition is described under the name and hence some confusion has entered the discussion of the subject. The typical hypertrophic stenosis is usually not present at birth if we are to judge by the time at which the symptoms develop. The characteristic manifestations, although absent at birth, appear during the first few weeks of life. At length a pyloric tumor may be palpable. This mass depends upon the enormous development of the muscle layers forming the walls of the pyloric canal which becomes a tube, the tumor-like extremity of which projects into the duodenum. The mucosa is thrown into longitudinal folds, which contribute in obstructing the canal, the ends of which project into the antrum pylori. There is found to be an increase in the number of muscle fibers, and the individual fibers are broader than usual. This hypertrophy occurs in the longitudinal layers although less extensively than in the circular layers. The reason for this remarkable hypertrophy has been accounted for on various grounds, but none of these are above criticism. admirable article on the subject by Cumston, the principal theories in explanation of the disease are given. The first includes a congenital malformation, a primary hyperplasia of the pylorus without the intervention of spasm; the second explanation would account for the muscular hypertrophy by spasm commencing during intra-uterine life and increasing after birth. The third theory would explain the hypertrophy as the natural result of simple pylorospasm, depending upon the usual causes and commencing after birth when nursing is begun,/thus explaining the hypertrophy as a secondary manifestation. It will be seen that the first hypothesis is that of a congenital malformation and not a little evidence has been found to support it. Whether or not it is possible to explain this remarkable affection purely on the ground of spasm, it is certain that the element of spasm ultimately plays an important rôle in its development. This would appear from the statements of Berend,2 who reports two cases occurring in his own family. Loreta's operation was done when the child was about one month old. It is noted that the surgeon felt the pylorus relax in his hand during the operation, although the thick muscle was still to be felt. was no narrowing whatever of the pyloric canal. case seems to prove beyond question the importance that spasm played in the trouble. It certainly has much to do with the development of symptoms even though it is not held responsible for the hypertrophy. In a thorough review of the subject by Fredet and Guillemot,3 there are collected and analyzed 598 published cases, in which

<sup>&</sup>lt;sup>1</sup> Interstate Med. Jour., Apr., 1911. <sup>2</sup> Jahrb. f. Kind. Heilk., 1910, LXXII, No. 2. <sup>3</sup> Congr. de Gyn. d'Obst. et de Ped., Sept., 1910.

there is shown a predisposition for the affection to occur in the Anglo-Saxon race, as compared with the Latin and Slavic people. Occasionally there appears to be a family predisposition to the disease and it occurs in boys more often than in girls. Congenital syphilis occasionally seems to bear some influence in the etiology.

Symptoms.—At birth the children are well nourished and usually conduct themselves like normal infants for a few days or weeks. Often at first, they develop remarkably, less often there is noted a delicate digestion from the beginning. The first symptom is vomiting, perhaps preceded by a few days of abundant regurgitation. The stomach empties itself rather violently after each nursing; the bowels become constipated, the urine scanty, and the patient greatly emaciated. Upon examination. in a goodly proportion of cases, a small movable mass may be palpated in the pyloric area and the stomach is sometimes found to be dilated. Marked peristaltic movements of the stomach are to be observed, passing obliquely from the left upward and to the right toward the pylorus. These constitute the important symptoms of pyloric stenosis of infancy. The tendency of the disease is to become progressively worse, anuria appears and the child succumbs from inanition.

Treatment.—There is a wide divergence of opinion as to the proper course to be adopted. Cumston says that he is satisfied that no case of true hypertrophic stenosis of infancy recovers without surgical intervention. On the other hand, R. Hutchinson, who appears to have had a large experience with the disease, says that in 17 out of 20 cases under his own observation treated at home, all recovered without operation. Three were sent to the hospital, where two died. A fair proportion of recoveries

<sup>&</sup>lt;sup>4</sup> Brit. Med. Jour., Oct. 8, 1910.

under medical treatment are reported by Huebner, Stark and others. The statistics of the operative treatment are usually unfair to the surgeon for the reason that most cases are not sent for operation until after medical treatment has been proved unavailing, and not until they are too enfeebled to safely undergo the shock of an operation. F. E. Bunts of Cleveland. who operated upon seven cases, four of which recovered, believes that the mortality would decline to 25 per cent or lower with early operation. After having carefully reviewed the reported cases, I am satisfied that in many of those which recovered under medical treatment, the disease was undoubtedly true hypertrophic pyloric stenosis, and not mere pyloric spasm, occurring in infants. I have not personally seen a sufficiently large number of cases to form a positive opinion as to whether medical or surgical treatment should be selected. If surgical assistance is to be invoked, the operation should be done early. In medical treatment we must be careful not to practice lavage too frequently, for little is gained thereby and a great deal lost. The stomach should be rested for a time and rectal alimentation attempted. Bismuth, oil and belladonna are the remedies that have done the most good. Alkalies are indicated in a few cases in which hyperchlorhydria is present; however over-acidity is not necessarily a feature.

<sup>&</sup>lt;sup>5</sup> Am. Jour. Med. Sci., Jan., 1912.

## CHAPTER XXXI

### VOMITING

We usually relate vomiting too exclusively to the stomach, forgetting the early lessons in physiology. fact, vomiting is a rather systemic and comprehensive act that implicates the organism. Appertaining primarily to the autonomic nervous system, it includes the activities of the sympathetic and the cerebrospinal systems. As an act of disgorgement it involves secretion, motion and sensation, calling to action vessels, nerves, muscles and glands of the head, trunk and extremities. The cold. dripping surface, the accelerated respiration and circulation, the active secretion from the salivary glands, eyes, naso-pharynx, tracheo-bronchial and urinary tracts, the straining muscles and the mental revulsion, are all testimonies of the somewhat universal nature of the performance.

Why recite these well-known facts? Because it emphasizes the use of recognizing the widely distributed radiations that result from irritation of the vomiting center; and it suggests the correlated notion that irritation at one or more of the peripheral points may start an impulse to the center, to be followed by a reflex to the stomach producing vomiting.

It would require a wider view than our present knowledge of physiology affords to explain fully the mechanism of vomiting with all its implications. We know that it results from various forms of encephalic irritation including the functional and psychic; not alone does this refer

to the special senses, touch, smell, taste, sight, hearing, but to ideation.

A large number of emetics owe their action to their toxic effects when conveyed to the center through the circulation.

In a general way, then, it may be said that vomiting results from local brain irritation, and from irritation carried to the center of emesis through the medium of the blood. It also results from certain remote irritations having a mode of action that requires additional explanation; for instance, vomiting that often accompanies the parturient act, that follows defecation in a few; that attends great pain and that accompanies appendicitis, renal calculus, etc., with absence of pain.

Finally, vomiting may be excited from certain somewhat closely associated efforts such as coughing, hiccoughing, clearing the throat. It is closely related to the act of regurgitation, more remotely to that of eructation which in the case of infants appears to be a natural method of relief from over-repletion.

Approximating infantile regurgitation is the practice of rumination.

Cerebral Vomiting.—The vomiting of central nervous disease is usually to be recognized by other concomitant symptoms. It is a well-known indication of cerebral pressure from syphilis, tumor, meningitis and apoplexy. Vomiting, as pointed out by Musser, may precede a stroke by hours.

Usually cerebral vomiting is unaccompanied by nausea. The vomitus is ejected unexpectedly, sometimes forcibly. In slowly growing brain tumors there is occasionally the history of recurring periods of vomiting without apparent cause for months before the appearance of other pressure symptoms. Also in chronic meningitis, espe-

cially tubercular, vomiting may anticipate the other symptoms.

Profound chlorosis may cause emesis that corresponds to central vomiting and is an indication of cerebral disturbance. Vomiting may result from irritation of the spinal cord, for instance, such as occurs in tabes dorsalis, which is considered in the chapter on Syphilis.

Vomiting from Gastric Irritation.—When emesis accompanies disease of the stomach it is usually secondary to local irritation. The familiar examples are gastric ulcer, cancer, pyloric stenosis, gastritis, and gastrectasis.

In certain functional diseases of the stomach we cannot thus readily decide upon the cause of vomiting. At times it is evident that the irritation which results from over-secretion, from hyperesthesia or from spasm, may excite enough local disturbance to induce vomiting; but usually there are other contributing causes at work. Any extrinsic trouble which may give rise to abnormalities of secretion, sensation or motion, may at the same time act directly upon the vomiting center thus favoring emesis. An illustration of this is the nausea and vomiting which arise in some instances of eye-strain. The same patient may suffer from vertiginous sensations, occasional attacks of headache and vomiting, which seem to have little relation to the rather continuous gastric disturbance which is present at the same time.

Uremic Vomiting.—Facts that closely correspond with those above stated also appertain to certain cases of toxic vomiting, for instance, in uremia. At times there is abundant proof of gastric irritation or even gastritis. Although ample local cause for vomiting often accompanies uremia, it will be found that there is a coexistent central brain irritation perhaps accompanied by cerebral

edema which would induce vomiting even without the irritated stomach.

It is a peculiarity of the vomiting of uremia that it may be temporarily the only symptom of intoxication. The urine may show no evidence of disease, there may be absence of edema, headache and diarrhea, but the vomiting persists, regardless of treatment of the stomach, until the toxemia is relieved by diet, purgatives and baths. Apparently in these cases there is the production of a toxic substance especially emetic in its action. The path leading to the result is not always the same, as illustrated in some types of vomiting of pregnancy; that is, what we class as uremia is not a definite entity, the manifestations of which may be forecasted. Its symptoms may depend merely upon renal insufficiency or may result from renal infiltration or degeneration; they may represent the effect of some general infection; they may be the expression of a defective blood supply, or may be related to the mischievous action of allergie. The vomiting of uremia may depend upon one or more of a variety of causes, some of them most obscure: the same may be said of vomiting in the general infections, such as the exanthemata and typhoid fever.

Reflex and Obstructive Vomiting.—In cholecystitis we do not know to what extent the vomiting comes from irritation transmitted to the stomach, how much from irritation of the nerve endings in the gall-bladder and how much from intoxication. Similar difficulties confront us in seeking an explanation of the vomiting of appendicitis, colitis, kink of a ureter, etc.

The vomiting of ileus or other forms of intestinal obstruction, includes among various factors those of derangement of the peristaltic rhythm and the advent of antiperistalsis. The latter is believed to cause fecal

vomiting. Fecal vomiting may be occasioned by hernia, internal and concealed, or external, but unsuspected, because of absence of local symptoms of strangulation. At times in obstruction, the harmony between secretion and absorption is broken, with the result that a remarkable amount of fluid, such as occurs in cholera nostras, continues to be ejected. An abundant vomiting of bile results from obstruction in the duodenum below the papilla or even lower in the small intestine. The incessant vomiting of green fluid in peritonitis, although characteristic, is yet not fully explained.

Vomiting from Eye-Strain.—In certain individuals, paroxysmal vomiting is occasioned by irregular and asymmetrical astigmatism and other forms of eye-strain. George M. Gould has taught that this may be recognized by instillation of a mydriatic in the eye sufficient to paralyze accommodation; following this the emesis is brought to a halt, probably because it excludes the action of the ocular branches of the autonomic nervous system.

Sympathetic Vomiting.—Among the more frequent causes of what may be termed associative vomiting is that which occurs in pertussis and in phthisis. This form of emesis is usually post-prandial in nature and introduces a special element of danger because of the resulting innutrition. Vomiting is of graver import occurring when gastro-enteritis is added to pertussis and laryngitis to pulmonary phthisis.

Vomiting from Metabolic Causes.—Although distinct from periodic vomiting (to be described later) there is a type of vomiting which recurs from time to time, often associated with headache. This form of vomiting resembles that produced by eye-strain, yet in a minority of cases it depends upon obscure metabolic derangements

usually related to the gouty diathesis and may be controlled by a régime of diet, bathing and exercise.

Post-traumatic and Post-operative Vomiting.—This, at times, appears to depend upon conditions that are allied to those producing at other times acute dilatation of the stomach. Such patients demand treatment for shock and must be closely watched lest dilatation develops undetected. At other times this form of vomiting apparently results from the direct effect of the anesthetic upon the vomiting center and in still other instances it, at least in part, results from excessive salivation excited by the anesthetic and the accumulation of saliva in the stomach. Vomiting of this latter type may be in a degree prevented by the practice of gastric lavage at the close of the anesthesia.

Treatment.—The control of vomiting involves the application of widely different principles, and, therefore, it is difficult to lay down definite rules of treatment. When emesis depends upon gastric irritability, it is necessary to give a period of complete rest to the stomach; meantime the demand for fluid may be satisfied by resort to enteroclysis. During the period of fasting ice should be applied to the epigastrium; preceding the fast it is sometimes advisable to wash out the stomach. When alimentation is resumed one should begin with a teaspoonful of toast water, rice water, infusion of raisins or thin arrow-root gruel, and should gradually increase the quantity. Experience teaches that liquid nourishment should be given either ice-cold or as hot as can be swallowed. When it is found possible to increase the quantity of aliment, it is sometimes advisable to substitute a hot fomentation for ice over the epigastrium. Gastric lavage, repeated even two or three times a day, is often indispensable.

Remedies having a locally sedative action are useful when the source of emesis lies in gastric irritation. The following prescriptions are among the most reliable:

R Cerii oxalatis	.30 .60	(gr. v.) (gr. x)
To be taken in a spoonful of water.		
Phenolis	.60	(m.x)
Aquae laurocerasiaa 6 One teaspoonful, p.r.n.	0.00	(3ii)
R Cocainae hydrochloratis	.50	(gr. ii) (m. viii) (3ii)

The wine of ipecac, also the wine of antimony, given in minute doses (one drop in a teaspoonful of water), have sometimes a happy effect in relieving nausea and vomiting.

When the source of vomiting is in central nervous irritation, remedies acting locally in the stomach are of little avail. Vomiting of this type may be controlled by bromids, chloral or veronal per rectum. To be effective relatively large doses are required. In central nervous vomiting, codein or morphin may prove to be the best remedy.

The suffering of mal de mer is sometimes obviated by taking a full dose of veronal just before sailing and by subsequently taking sufficient amounts to calm the vestibular nerves. Strontium bromid has a similar effect, is less toxic and for long voyages it is preferable.

Occasionally champagne, given frappé, is more effective than sedatives, but is not advised when there is irritation of the gastric mucosa or toxemia.

Depending upon toxic causes, vomiting is rarely overcome except after free elimination. A calomel purge or a hot pack are here more successful than stimulants, sedatives or anti-emetics.

In the post-prandial vomiting of phthisis relief may attend the administration immediately after food of menthol in doses of .03 (gr. ½) given in a sherry-glassful of some pleasing emulsion. This may need to be followed up for several days before the full effect is obtained. Cannabis indica in rather full doses is also of service.

Vomiting is sometimes largely dependent upon autosuggestion and should be dealt with by psychotherapy. It is a mistake to suppose that all patients ought to be confined to a liquid diet. Although this is desirable in some cases, it is objectionable in others. Although a complete fast for twenty-four or forty-eight hours is often indicated, too prolonged fasting induces acidosis. It is imprudent to impose a prolonged fast without the use of enteroclysis and the introduction of alkalies. The advantage of a fasting period is that it rests the excitable pneumogastric reflex or permits improvement of an irritable mucosa.

Vomiting of Pregnancy.—This should be divided into:

- (1) Vomiting that occurs during pregnancy, not dependent upon it.
- (2) Vomiting that results from the pregnant state, that is, in some way depends upon the contingencies of the pregnant state.

The former may arise from any one of a great variety of causes which are exaggerated during pregnancy. In this type there is usually a pronounced disturbance of the digestive tract and often the vomiting may be largely relieved by dietetic measures. Associated with it is intestinal auto-intoxication, functional disturbance of the liver and renal inadequacy.

The various forms of reflex vomiting, such as that following eye-strain, are often increased during gestation.

The second type, the true vomiting of pregnancy, is represented by different forms. In one the trouble seems to be reflex and to depend upon the unusual stress going on in the uterus. It may be promptly relieved by moderate dilation of the cervix uteri, a maneuver that surely would have little effect upon auto-intoxication.

A second group of cases seems to result from sapremia, and may be relieved by dietetic restriction and by increasing elimination.

(3) In the third group belong the obstinate, incoercible cases, apparently the result of some special intoxication, the origin of which is debatable. known toxin gives rise especially to parenchymatous degeneration and necrosis of the hepatic cells. Stone and others, who have emphasized this fact, call attention to the diagnostic importance of an excess of ammonia in the urine. It is understood that in protracted vomiting and starvation, no matter what the cause, the excess in urine ammonia is to be expected, as a natural antagonism to acidosis. Recently Underhill and Rand 1 announced the belief that an excessive ammonia output in pernicious vomiting should not necessarily be ascribed to lesions in the liver, but rather to the starvation depending upon vomiting. All the manifestations of acidosis occur in these cases and may be suspected by the odor of acetone in the breath. Diacetic and oxybutyric acids occur in the urine, often associated with albumin, at least in traces, and with renal epithelium, tube casts and blood cells. It does not appear that the conclusions of Williams as to the causative effect of hepatic necrosis in the pernicious vomiting of pregnancy, have been disproved.

<sup>&</sup>lt;sup>1</sup> Arch. Int. Med., LXI, 1910.

The necrosis of liver cells is sometimes extreme, with the development of what we have long known as acute yellow atrophy of the liver. Apparently there are cases with marked metabolic disturbances dependent upon starvation, in which the intoxication may be relieved and the acidosis controlled by introducing carbohydrates, thus increasing the supply of glycogen to the tissues. The administration of sodium bicarbonate is also demanded. These measures, however, would appear to have little effect upon these cases with well-developed necrosis referred to by Williams.

The not unreasonable theory has been advanced that the special toxic substance, present in hyperemesis gravidarum, develops in the fetus and is absorbed by the circulation of the mother. This would offer a satisfactory explanation of the fact that these dangerous cases may be promptly relieved by the timely interruption of pregnancy. Too long delayed, this measure proves unavailing because of the parenchymatous changes to which the intoxication has already given rise.

Hysterical conditions in pregnancy may be attended with vomiting that is misleading and alarming, as illustrated in the case reported by M. Potocki.<sup>2</sup> A woman of 24 in a previous pregnancy had such intractable vomiting that abortion was performed in the second month. She again became pregnant and at the third month of gestation the vomiting appeared as before. The pulse was 140, the temperature 37.4° (99.3° F.), yet the patient had not the general appearance of auto-intoxication; rather, the nervous symptoms were striking. There was great agitation, talkativeness, etc., and the patient demanded that she be aborted as before. She showed emaciation, a subicteric appearance, increased salivation, yet

<sup>&</sup>lt;sup>2</sup> Bull. Méd., Jan. 15, 1910.

neither fetid breath nor buccal ulceration. Moreover, urine was abundant and of good quality. The liver and spleen were rather small, and the blood pressure was 150. The physician, after making an impressive examination, informed the patient that she was not pregnant, that her vomiting was simply nervous, and was rewarded for his mendacity by finding the condition of his patient ameliorated on the following day. A complete recovery with normal delivery ensued.

There are relatively tractable forms of hyperemesis in pregnancy, arising from hysteria, reflex excitability, etc., as well as from special and threatening forms of intoxication, which are susceptible of control without abortion. However, delay in resorting to this measure in vomiting of the third type is disastrous, except in those rare cases in which prompt improvement follows medical treatment. The over-conservative course means the loss both of mother and child.

TREATMENT.—Early in pregnancy the urine should receive special study, for which purpose "twenty-four hour specimens" should be examined frequently. the decline of urinary solids the rise in total acidity and the increase of ammonia, there will develop noticeable symptoms of intoxication previous to the onset of vomiting. Upon the appearance of the symptoms of renal insufficiency, the diet should be restricted, animal proteids and other foods rich in purin bodies should be excluded. Purgatives, colonic irrigation and cabinet bath are indicated. Should the vomiting persist, gastric lavage should be practiced. The acidosis may be relieved by the repeated introduction per rectum of a solution of sodium bicarbonate 8 gm. (3ii) in 500 c.c. (Oi) of water. The patient should be given complete rest in a shaded room with special attention to ventilation,

In milder cases these measures may be somewhat relaxed, but in all pregnant women the urine should be frequently examined for indications demanding a restricted diet and special measures of elimination.

#### CYCLIC OR RECURRING VOMITING

This term was first employed by Leyden to describe a periodic occurrence of vomiting, gastralgia and prostration. His cases showing vomiting, gastralgia and nervous excitement resembled the "gastric crises" depending upon tabes, but were noted for their somewhat definite periodicity. (See Gastric Syphilis; also Gastrosuccorrhea Periodica.) He thought that the condition might depend upon neuralgia of the vagus or celiac plexus, and that there was absence of structural disease of the nervous centers.

Recurrent vomiting of a similar type may arise in irritation of the autonomic nervous system from disease in the abdomen and elsewhere. Hence it may depend upon latent appendicitis, cecal stasis, gall-bladder irritation, chronic pancreatitis and movable kidney; or upon eyestrain, disease of the labyrinth, chronic meningitis or other central trouble. Examples of each of these have come under my own observation.

Often it is difficult to exclude the recurring intoxications, as uremia or spinal cord disease (tabes). The vomiting from brain tumor and other forms of brain irritation is not usually accompanied by gastralgia, is unheralded by premonitory symptoms and is sudden and often projectile. There is a type of recurrent vomiting associated with general metabolic depression in which there is seen the picture of auto-intoxication with acidosis. These cases differ from the periodic migraine or sick headaches, and from those which occur with each

menstrual epoch, in the following respects: (1) headache is not necessarily a feature; (2) the metabolic disturbance is more striking, and (3) the attacks persist longer than is usually true of sick headaches, although there is much irregularity in duration. Gastralgia may or may not be present. There is usually a short premonitory period of unrest and digestive disturbance.

In hysteria vomiting with or without pain sometimes recurs periodically, often at the time of the catamenia. It is accompanied by the characteristic stigmata which stamp its hysterical nature.

Angioneurotic edema, first described by Quincke and to which Osler has directed especial attention, may attack the stomach, produce pain and symptoms in other respects, not to be differentiated easily from periodic vomiting.

Arteriosclerosis and the vascular crises of Pel may explain certain cases, yet in these the element of pain is dominant.

Only when the vomiting is so persistent as to obscure other features of the case and thus distract the examiner's attention is there a probablity of mistaking for cyclic vomiting that which accompanies gastric or duodenal ulcer; yet with a singular hiding of the other symptoms, vomiting of a recurrent type may be occasioned by peptic ulcer. None of these are indentical with the true cyclic vomiting of children, which disease deserves careful consideration.

Cyclic Vomiting of Childhood.—The symptoms composing this complex are persistent vomiting, great prostration, thirst and nervous depression, usually excessive secretion of gastric juice, a decrease in the urinary secretion, generally constipation, but occasionally diarrhea, acidosis, acetonemia, a disturbance of pulse rate, usually

increased, sometimes retarded and a disturbance of temperature, sometimes marked hyperthermia but occasionally hypothermia.

Case No. XXXIII.—I studied the subject for the first time in a child three years old who did not become exempt from the recurrences until he had reached his thirteenth year. The attacks came on with some regularity at intervals of from three months to six months. First there was languor and loss of appetite. The child became very nervous and fretful, yet his complaint was rather indefinite. There was a slight elevation of temperature which usually preceded the vomiting. The material ejected was a thin, watery mucus which had all the characteristics of gastric juice. The bowels were constipated, but responded to the use of calomel and magnesium sulphate. The urine was diminished in quantity, highly acid in reaction and in the sediment were many uric acid crystals. After a time acetone and diacetic acid were present. During the height of the attack the child was profoundly depressed but nervously irritable; his immediate dissolution was at times feared. Each attack continued from three to five days, when convalescence ensued definitely and rapidly. As is the rule, this child was of a somewhat neurotic type, yet not sickly; he was active and happy when free from his attacks. scribes a typical case, and although variations are seen. the affection is sufficiently characteristic to make its recognition easy. The disease is less likely to attack infants than children two or three years old, and the paroxysms cease to appear at the age of puberty or shortly before that. Following their disappearance, the patient may exhibit no abnormalities, although a subsequent history of asthma is present in some cases. the group of cases reported by Griffith and others before

the Association of American Physicians in 1900, a number terminated fatally and one such result has come under my observation.

Cyclic Vomiting.—Comby 3 reports 72 cases of cyclic vomiting in which he found a family predisposition in a There were 7 cases in three famicertain proportion. He believes that 30 per cent of his cases have been occasioned by enterocolitis; constipation was present in 80 per cent, with or without colitis, and in 18 per cent there existed symptoms of appendicitis. These statements lead one to feel that cases other than cyclic vomiting have been included under this head. Commenting on these cases Marfan points out that, given the proper condition, a variety of accidental causes may precipitate an attack of cyclic vomiting accompanied, he believes, by a state of acetonemia. He does not pretend to know why the excess of acetone appears, but he affirms that it is an integral part of the attacks of cyclic vomiting. Other French observers are of the opinion that acetonemia is merely the result of intestinal fermentation. The latter condition has not been recognized as belonging to cyclic vomiting, while acetonuria is nearly constant in prolonged vomiting, or fasting, from any cause. Nor has indicanuria, indicative of intestinal putrefaction, been a marked symptom in these cases.

Morse regards acidosis as a cause of the syndrome. Snow believes that the vomiting is the result of intermittent hyperchlorhydria and that the latter is an expression of a neurosis.

Ely would look upon the disease as the expression of perverted metabolism, the result of a toxic substance belonging to the alloxur series.

I think that the conception of the disease should be

<sup>&</sup>lt;sup>8</sup> Bull. Méd., Dec. 22, 1906,

limited to the symptom complex, which I have described. for in children, as well as in adults, occasional attacks of vomiting with prostration may depend upon a variety of causes which bear no necessary relation to the disease in question, and yet which may be mistaken for it. With Dr. DeWitt Sherman I have studied a child of nine who for two years has had recurring attacks of vomiting, prostration and slight temperature disturbance. case might have been mistaken for cyclic vomiting but for the appearance of glycosuria. This led to the precise study of the child's metabolism which showed that the symptoms were metabolic in origin, and that indiscretion in diet, as well as nervous excitement or physical tire, were sufficient to produce the attack. In my cases there has uniformly been present, during the seizures, an excessive secretion of highly acid gastric juice. acidosis is probably, in part or in whole, not the cause but rather the result of the vomiting and the consequent denutrition. The condition impresses me as being neurotic in nature. In two of my cases there was found to be eye-strain, as the result of refractive defects, and upon the correction of these the attacks disappeared. In one case when the child's glasses were not worn for two days the symptoms developed, but with greater attention to the constant wearing of the lenses the child remained well. As some of my colleagues have had experience analogous to this, I am led to attribute considerable importance to eye-strain as an etiologic factor. The condition may have a vagotonic basis.

Treatment.—In the very beginning of the attacks a full dose of calomel, followed by a saline, appears to lessen their intensity. Considering the toxemia which sometimes develops, this is at least a rational measure. One case seemed benefited by rather frequent small doses

of liquor magnesii citratis. Gastric sedatives and antacids are of little avail. When the depression is profound enteroclysis is indicated. Griffith reports favorably on the effect of morphin hypodermically. In some of his cases the good effect was remarkable, but while the drug is at times indispensable it can hardly be accepted as a routine measure. Atropin has not succeeded as well as might be expected, perhaps because not usually given in sufficiently large doses. Hot packs every four or six hours have a favorable effect. When they are given care should be taken to move the patient with the least disturbance possible.

Periodic vomiting in adult life is produced by such a variety of causes that to lay down specific lines of treatment is fruitless.

Among the general principles an important one is fasting until the attack subsides. During fasting rectal alimentation and enteroclysis with a solution of sodium bicarbonate, (1 per cent), should be used with the view of controlling the acidosis, which is inevitably present during fasting and vomiting.

# CHAPTER XXXII

## ABDOMINAL PAIN AND TENDERNESS

Source and Character.—Abdominal pain and tenderness occupy the attention of both patient and physician and are looked upon as indicating disease. We have learned that these symptoms are not invariable and therefore much effort has been given to their exact interpretation.

That the abdomen is a region of peculiar sensitiveness is felt instinctively, as shown by the involuntary efforts made to guard it from injury. There is a noticeable difference in individuals as to the sensitiveness of the part, and some are so "ticklish" that it is difficult to discriminate between physiological and pathological tenderness to pressure or touch. There is less difficulty in estimating the importance of abdominal pain than tenderness. and yet even this is surrounded with confessed possibilities of misapprehension. It is generally believed that pain is felt especially in the region where the irritation is located. McKenzie and Head in their careful studies show that the seat of pain has merely an indirect value in the location of the disease producing it. They hold that the pain is reflex, superficial, and that it is referred to a given area along certain nerve paths springing from a spinal segment, which is itself irritated from disease in some perhaps distant organ, which transmits impulses to its corresponding spinal segment. According to them the pain experienced is not necessarily located over the affected organ, but such may or may not be the case. Thus the pain from stenosis of the coronary vessels of the heart may be experienced in the left shoulder and arm, and the pain of a diseased gall-bladder may be felt in the back and right shoulder. While not venturing to disregard the principles of this teaching, I agree with Cabot that it does not afford a sufficiently reliable means of information as to the location of the source of most abdominal pain. It is well recognized that the pain of visceral disease is not always located directly over the affected organ. Many observers contend that the pain is not seated in the organ itself, but in the abdominal parietes corresponding thereto, and especially in the peritoneum.

Lennander holds that the pain in visceral disease, for instance ileus, does not arise from the intestine, innervated only by the sympathetics and vagi, which lack sensory fibers; but from the abdominal wall, which is innervated by the cerebrospinal nerves. Also he believes that there may be painful adhesions having spinal innervation, formed between the abdominal wall and the stomach or intestine. Mouthin, who attributes the pain of gastric ulcer to irritation of the parietal peritoneum, calls attention to the fact that the lymph vessels of the gastric walls extend to the parietal peritoneum, and hence provide a special physical call for sensitiveness there.

Riedel considers epigastric pain to be (1) reflex; (2) transmitted from some neighboring organ, and (3) local, as in peptic ulcer. Of the important reflex causes he includes appendicitis, epigastric hernia, scars of old ulcers, and irritation in the transverse colon; and as instances of pain transmitted from the neighboring organs, he names that of gall-bladder affections, movable kidneys, and pancreatitis. The transmitted pains he considers are usually located on the right side, and he regards left-sided pain as a valuable diagnostic sign of gastric ulcer.

An attempt to distinguish between reflex and transmitted pain is confusing; how may we know that so-called transmitted pain arising in abdominal disease is not reflex? We recognize local pain and pain which is felt at some distance from the seat of trouble. It is not always possible to say whether it is a transmitted or a reflex pain, although it is admitted that both explanations are tenable.

Transferred pain without local or reflex cause may be illustrated by the pain of the gastric crises of tabes dorsalis. Another example is the abdominal substitute of angina pectoris and the epigastric agony of rupture of the left ventricle. A good instance of reflex pain is that experienced in a zone along the outer aspect of one thigh, occasioned by irritation from a calculus in the corresponding kidney, and quite unlike the pain in the same case felt along the genito-urinary tract, which pain may be transmitted. The term "referred pain" is often used comprehensively, and embraces both transmitted and reflex pain.

In the abdomen it is not always easy to decide whether a given pain should be classified as reflex or transmitted. It is not yet decided whether local pain in the viscera is properly so classed. For instance, the pain of peptic ulcer is usually definitely limited to a small epigastric area near the median line a short distance below the xyphoid, regardless of the location of the ulcer or the position of the stomach. Experimental research appears unable to show that sensory impulses of any kind are conveyed by the splanchnics from the gastric mucosa; and evidence is offered that this structure is itself lacking in sensation.

<sup>&</sup>lt;sup>1</sup> F. R. Miller: "Visceral Sensation, etc.," Inaugural Dissertation, Münich, 1911.

We are in need of further light on many sides of this important subject and for the present must be governed by what clinical experience seems to indicate. In this instance it leads us to regard the pain of gastric irritation as local in character, yet even this form of pain may turn out to be reflex.

A very misleading example is the abdominal pain, variously located, which depends upon incomplete and unsuspected inguinal hernia. A group of such cases I reported in *Transactions of American Physicians*, 1904. This pain probably depends upon tension of the hernial sac communicated to the peritoneum. It may be recognized by relief which follows proper support from a truss.

Abdominal pain is often heightened by increase of local or general intra-abdominal pressure, and hence becomes more manifest with motion, effort, upon assuming

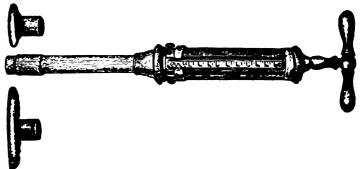


Fig. 57.—The Algesimeter of Boas.
(From Dr. Hans Elsner. Lehrbuch der Magenkranheiten; Berlin, 1909.
Verlag von S. Karger, p. 30.)

the upright position or certain other postures. In studying the effect of pressure, there is sometimes advantage in using the algesimeter of Boas.

Schmidt points out that the lateral lying posture most often increases abdominal pain, which is less noticeable

when lying on the back, as is well illustrated in peritonitis.

Various Pathological Conditions.—Pain of ulcer may be suddenly increased because of gastric distension or overtonicity and may as suddenly subside with the disappear-The same principle applies to pain ance of this tension. in the inflamed gall-bladder or appendix. On the other hand merely sympathetic or neuralgic pain is often relieved by firm pressure. In a recent address, Crile 2 affirms that abdominal visceral pain is caused by tension of the muscular wall of the part and not by irritation of the mucosa. He points out that cutting of the mucous membrane causes no pain, while stretching or tension of the walls of the organ produces pain. Thus the pain of appendicitis, cholecystitis or peptic ulcer would depend upon over-tonicity and tension of the walls of the affected Substantiation of this statement, I conceive, may be found in the following: Physiologists have discov-. ered that subcutaneous injections of adrenalin solution overcome the tonicity of the stomach or intestine. Overtonicity of the stomach accompanying gastric ulcer, etc., is accompanied by pain, sometimes violent. With a view of testing clinically the statement of the physiologists. I have injected adrenalin solution in these cases with the result of relieving the pain instantly.

The pain of ilio-sacral disease is sometimes felt in the lower abdomen. This is commonly ignored owing to more intense and persistent pain in the back and in the sciatic nerves. The abdominal pain is rarely severe, yet in diagnosis it is worthy of consideration.

The gall-bladder when irritated, is an example of an organ which gives rise not only to referred, but to local pain and tenderness. Although the pain is usually epi-

<sup>&</sup>lt;sup>2</sup> Louisville Month. Jour. of Med. and Surg., 1912.

gastric in the beginning, it soon shifts to the gall-bladder, where local tenderness to pressure is elicited at a point where a straight line drawn from the right nipple to the umbilicus crosses the free costal border or just beneath this point. Tenderness is also experienced an inch or two from the spine over the tenth, eleventh or twelfth right rib (Boas). While pain may be referred to this

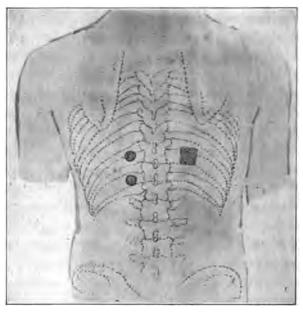


FIG. 58.—BOAS' POINT, REPRESENTED BY SHADED SQUARE AT RIGHT OF VERTEBRAE. Tender points of gastric ulcer, represented by shaded circles at left of vertebrae.

point, it often radiates upward to the right chest and shoulder but rarely extends downward. In many cases the pain is constantly referred to the epigastrium.

The pain of renal calculus or movable kidney may occur either in the region of the disease, or at the epigastrium or more particularly, down the genito-urinary tract. In the female more often than the male, diseases of the pelvic organs may give rise to pain in the abdomen. Previous to the appearance of the eruption of herpes zoster, pain is experienced often anteriorly along the course of the spinal nerves. The accompanying localized tenderness and leucocytosis may lead one to suspect appendicitis, or inflammation in other viscera.

The angor abdominalis, depending upon inflammation of the abdominal aorta or its branches, is severe and generally misinterpreted. It is accompanied by tenderness along the abdominal aorta, which vessel, owing to thickening of its walls or to periaortitis, seems upon palpation to be broader, more resistant and more irregular than normal. The condition has been described ably by J. Teissier of Lyons. It may be of gouty or infectious origin and may then be amenable to treatment, in which it differs from chronic arteriosclerosis. In the latter the cause persists, and although the pain may be relieved it recurs and usually is accompanied by other evidence of degeneration. The paroxysms are often excited by physical efforts or functional stress.

Pains analogous to the above arise, when arteries are but slightly diseased from the angiospasm or vascular crises described by Pel. Any of these arterial conditions may relate definitely to the stomach. The subject of vascular spasm has been discussed by Barker, who refers to the high blood pressure which we should expect to accompany it. Barker also draws attention to the occurrence of high blood pressure in tabetic gastric crises and lead colic. The rise in blood pressure attending attacks of lead colic is not invariable. Schmidt, in saying that the phenomenon is largely a question of vasomotor control, relates cases in which the pressure fell during the attack.

Lead colic, as Cabot states, is notorious in this that, though the toxic cause is widespread, showing itself in diffuse abdominal pain, it may signal its presence by intense pain located at a strictly circumscribed point.

Angioneurotic edema affecting the abdominal viscera may account for localized pain, tenderness and rigidity counterfeiting inflammatory processes.

Epigastric hernia, occasionally so small that it escapes notice, may produce pain identical to that of gastric ulcer, and in certain instances there is supposed to be an obscure relation between these conditions.

A diseased pancreas gives rise to pain, localized just above the umbilicus. The pain of pancreatic calculus may extend somewhat to the right and might be mistaken for that of biliary colic. Chronic pancreatitis may give rise to paroxysmal pain and vomiting, usually with comparatively little tendernss. The pain of acute pancreatitis is limited to the region of the pancreas and In intensity it is variable; it may be agonizing. However, it is controlled by a smaller dosage of morphin than is the pain of hepatic or renal colic, for which it is sometimes mistaken. The accompanying symptoms are more suggestive of perforation or strangulation than of calculus disease. Cancer of the pancreas, when the disease extends upward, may give rise to pain and tenderness in the epigastric region. These sensations are sometimes modified (increased or diminished) by taking food and, in character, may be mistaken for the sensory symptoms of carcinoma ventriculi.

The pain of localized peritonitis while usually intense is at other times moderate in degree. This is especially true of tubercular peritonitis. The notable cutaneous hyperesthesia, the exquisite tenderness to pressure, the decubitus and the exaggerated tonus of the abdomen serve to characterize the pain of acute peritonitis.

A source of pain and tenderness just below the ensi-

form may be found in a dilated and distended right heart.

The tension produced by passive congestion of the liver in broken cardiac compensation also gives rise to pain and tenderness under the costal arch.

The following differences may serve to distinguish between them. In dilated right heart the tenderness is most marked high in the epigastrium, just below and under the ensiform, and may occur when the liver is not large. The pain and tenderness of congested liver are experienced over a larger area and are relieved by suitable purgation. The question of differentiation is often a fine one; yet, as the ultimate cause rests in failure in circulation in both instances, the indications for treatment are nearly identical.

Abdominal pain and tenderness, at times accompanied by rigidity, apparent tumor, tympany and leucocytosis, are well recognized symptoms of thoracic disease. Such pain may anticipate for days the physical signs in the chest. This unexpected manifestation occurs especially in pneumonia involving the lower lobe and pleuritis of the lower thorax and, occasionally, in pericarditis. Repeatedly I have seen severe abdominal pain in the right hypochondrium and the right iliac fossa depending upon thoracic disease masquerading as cholecystitis or appendicitis. Instructive examples have been described by DeLancey Rochester, also by Allan A. Jones.<sup>3</sup> The explanation offered for this manifestation is irritation of the lower intercostal nerves, branches of which are distributed to the abdominal parietes.

Appendicitis, in which the local pain and tenderness are so often diagnostic, may give rise to pain in various parts of the abdomen and, owing to malposition of the

<sup>&</sup>lt;sup>8</sup> Jour. Am. Med. Assoc., July 13, 1907.

appendix, the tenderness may be located remote from McBurney's point and be present at several other points hereafter to be indicated. The character of pain differs strikingly, depending at times upon colonic spasm and again on spasm or varied structural changes in the appendix, again upon local peritonitis or upon adhesions.

Affections of the colon may occasion abdominal pain or tenderness varying widely in character, intensity and location. Included are spasmodic colic from constipation and other causes, cecal stasis, cecummobile, diverticulitis, lead poisoning, uremic crises and other intoxications, besides enteroptosis, pericolitis and colitis. In one case of severe epigastric pain located in the right upper quadrant, upon laparotomy the cause was found to be adenocarcinoma of the ascending colon. So, also, inflammatory and fibroid tumors of the colon may occasion local pain and tenderness.

The pain of mucous colitis or, as von Noorden prefers to call it, "colica mucosa," may be intense and deceptive. It is well to recall Glénard's early description. Among its confusing characteristics is that of localization frequently accompanied by induration and deep tenderness dependent upon colonic spasm. There is greater embarrassment for the reason that mucous colitis may have its source in chronic appendicitis or other local dis-It is a mistake to insist that this is invariably the ease. Individuals may suffer from an inherent sensicase. tiveness of the colon; usually there is enteroptosis; occasionally no definite cause is discovered. Mucous colitis is to be recognized by patient examinations of the stools and by noting the relation that exists between defecation and the abdominal symptoms.

Cancer of the stomach may cause atrocious pain rarely referred; yet in some cases pain is conspicuously absent.

All degrees of gastric discomfort may accompany cancer, so that the symptom of pain must not be counted as diagnostic of the disease.

Gastric spasm, produced from any cause whatever, may be accompanied by pain, usually cramp-like in nature.

As before stated, I have been able to relieve this by the subcutaneous injection of adrenalin solution. This has served to verify clinically the statement of the physiologists and also to clinch the diagnosis of the cause of pain. This leads to the consideration of hyperchlorhydria and peptic ulcer.

Pain is often associated with hyperchlorhydria. just what extent this depends upon the superacidity is a question. I agree with Kaufmann that some other element in addition to the high acidity is probably concerned. Undoubtedly, when hyperacidity is secondary to peptic ulcer, fissure or other structural causes of pyloric spasm, the excess of hydrochloric acid sufficiently aggravates the trouble to produce pain. In the "gastric crises" of tabes, often accompanied by hyperchlorhydria, the pain is undoubtedly of spinal origin. The pain of gastrosuccorrhea periodica cannot be thus explained. the routine practice of stomach clinics, among the many cases of hyperchlorhydria, some are without pain while with others it is present in varied degrees of intensity. In many of these no explanation is found in accompanying structural disease. Perhaps the pain is associated with the function of internal secretion, as it certainly is with the state of over-tonus. At any rate it must be admitted that pain with slight tenderness occurs in cases which still have to be classed as functional hyperchlorhvdria.

In peptic ulcer pain is a common symptom. When the ulcer is at the cardia, the pain is experienced at the time

of eating. When the ulcer is located in the body of the stomach, the pain appears soon after the repast; when at the pylorus or duodenum, the pain is usually postponed, sometimes as much as three or four hours after meals. This is far from invariable, and the so-called "hunger pain," which is commonly attributed to duodenal ulcer, lacks the significance with which it has been credited. may be absent. It is true that "hunger pain" often appears with duodenal ulcer; but pain having precisely the same characteristics may be developed in pyloric ulcer, fissure, disease of the gall-bladder, appendicitis and other intestinal affections, as well as in duodenal ulcer. deed, in not a few cases of simple hyperchlorhydria, the pain occurs so late after meals that it cannot be distinguished from that of duodenal ulcer. The term "hunger pain" may be misleading. It has no necessary relation to appetite or hunger; it simply means that it occurs at a time when the stomach is emptying itself and that it is relieved by the taking of food; therefore it is not a question of hunger, but a question of pain relieved by eating which has given rise to the term "hunger pain." It is not a rare experience to find gastric pain relieved by the taking of food, and this occurs in a variety of troubles. Recently the subject has been wisely discussed by Allan A. Jones.4

Pain depending upon acute inflammation in any region is usually increased by eating, for instance in furunculosis.

As to the digestive tract, the ingestion of food may excite pain through the action of any one of a variety of factors.

Dr. Howard A. Kelly in an enlightening article on abdominal pain presents a useful diagram for the topo-

<sup>&</sup>lt;sup>4</sup> Jour. Am. Med. Assoc., 1912. <sup>5</sup> Interstate Med. Jour., 1911.

graphical marking of the usual seat of pain in varied affections. (Fig. 59.) He recommends the use of some such diagrams for recording and comparing the location of pain in the study of abdominal disease and also makes the useful suggestion that, when possible, the cause and location of the pain should be determined by deliberate attempts to reproduce it. For instance, by pressing deeply upon the kidneys or by handling bimanually the

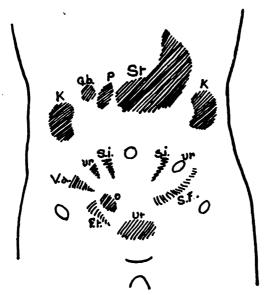


Fig. 59.—K, Kidneys; Gb., Gall-bladder; P, Pylorus; St, Stomach; Ur, Ureters;; V. A., Vermiform Appendix; O, Ovary; F. T., Fallopian Tubes; S. F., Sigmoid Flexure; Ut, Uterus. After Dr. Howard A. Kelly, by permission.

uterus, ovary, ureter, etc. In this connection it should be said that the pain of hyperchlorhydria or peptic ulcer may be promptly reëxcited by administering a relatively large dose of some mineral acid; and conversely, in case of gastric ulcer, the pain, if already present, may be relieved promptly by giving a gram of orthoform stirred in a little water, a useful recommendation of the late Dr.

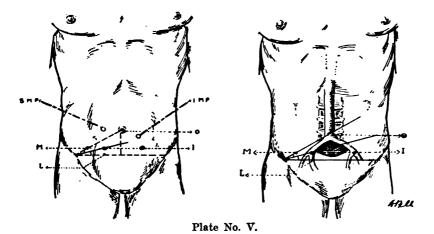
Murdock of Pittsburg. The precise location of pain is a matter worthy of more definite study than it usually receives, as is also the location of abdominal tenderness.

A useful contribution to the subject was made by Loeper and Esmonet.<sup>6</sup> They direct special attention to the painful points located symmetrically on either side corresponding with the branching of the iliac artery. They also call attention to the point of Lang in its relation to McBurney's point, the point of iliac tenderness and to the superior and inferior mesenteric points. Their instructive diagrams are here reproduced. These do not indicate the sensitive mesenteric points which I have taken the liberty of marking. Robert T. Morris has also made a valuable contribution to the study of the points of tenderness present in abdominal disease. To appreciate its significance one should consult the original articles.7

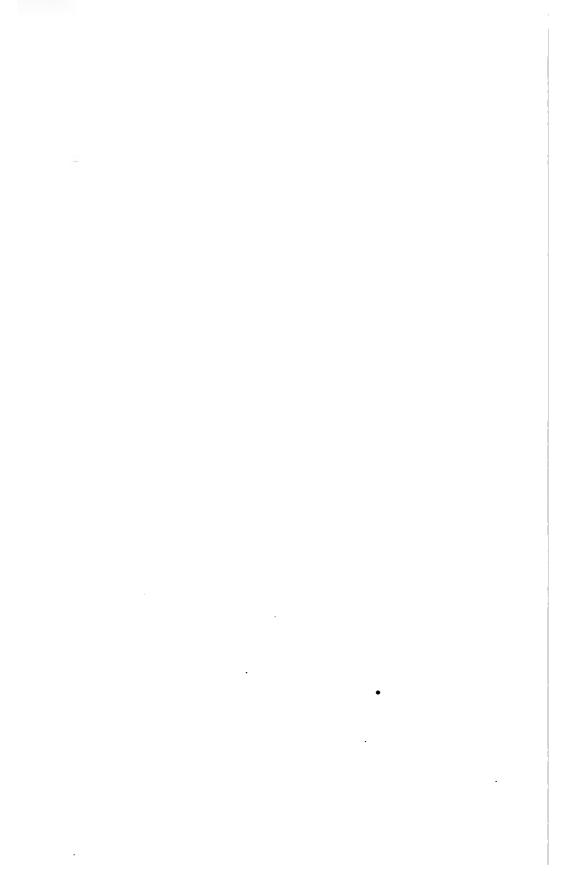
The point of McBurney is located at the junction of the external and middle third of a straight line uniting the umbilicus to the right anterior superior iliac spine. point of Lang is situated on the right side at the junction of the external and middle third of a straight line connecting the anterior superior iliac spines of the two The iliac points described by Loeper and Esmonet are symmetrical, but so far as concerns the right side, the point is a little higher than the point of Lang, slightly lower down than the point of McBurney, and is on a line which joins the anterior superior spine of the ilium with the tuberosity of the ilium of the opposite side, and about 4 cm. from the median raphe.

The mesenteric points are located over and correspond respectively with, the superior and inferior mesenteric

<sup>&</sup>lt;sup>6</sup> Bull. Méd., May 28, 1910. <sup>7</sup> Jour. Am. Med. Assoc., Jan. 25, 1908.



S. M. P., Superior mesenteric point; I. M. P., Inferior mesenteric point; O., Umbilicus; M., McBurney's point; L., Lange's point; I., Right and left iliac points. After M. Loeper and M. Esmonet, by permission. (S. M. P. and I. M. P. added by the author.)



arteries; on the right side nearly on a level with the umbilicus and one or two inches therefrom; on the left side one or two inches from the linea alba and about one inch below the umbilicus. These correspond closely with the points described by Morris, and already referred to.

Various Conditions.—Enteroclitis, gastritis and other inflammatory processes in the abdomen and pelvis, besides severe intoxications, may be accompanied by tenderness over the mesenteric and over the iliac points. Tenderness will be found at these locations in appendicitis as well as at the characteristic McBurney's point. Continental observers have insisted that tenderness at the point of Lang is even more indicative of appendicitis than that located over McBurney's point; however, most clinicians continue to value the diagnostic importance of the latter.

In the gastric neuroses, as well as in other conditions which excite the nervous system, it is usual to find marked hyperesthesia over the sympathetic mesenteric points. Therefore, in hysteria or in hyperesthesia from any cause, sensitiveness, sometimes exquisite, may be expected at these locations. Tenderness under like conditions, may be elicited by deep pressure along either side of the abdominal aorta where sympathetic nerve ganglia are richly distributed.

## CHAPTER XXXIII

### THE ESOPHAGUS

Structure and Function.—The anatomist may be interested in knowing that the esophagus is about nine inches in length, but the clinician values more the fact that in the average healthy adult the cardiac end is about sixteen inches from the incisor teeth, but that under pathological conditions the esophagus may be somewhat elongated.

The physiologist knows that in the passive state the esophagus is a collapsible, distensible, musculomembranous tube, having, when filled, a caliber of about 2 cm., slightly narrower at either extremity at the cricoid and at the cardia. The clinician is aware that it is also a dilatable tube and that it may, like other passages, suffer constriction, ectasia, or diverticulation. anatomist points out that the esophagus occupies the posterior compartment of the mediastinum and that it is in relation anteriorly with the loose tissue encompassing the trachea and the left bronchus, the left carotid and the arch of the aorta; it lies along the right side of the aorta and passes in front of it just before entering the abdomen. The pericardium and the left pneumogastric nerve are in front, the pleurae on either side, the right pneumogastric behind. Posteriorly it rests upon the vertebral column. In the neck it is closely related to the thyroid gland and to the recurrent laryngeal nerves.

The clinician remembers that in dealing with disease of the esophagus he must be mindful that a morbid proc-

ess involving these neighboring structures may directly invade the esophagus or indirectly disturb its function just as disease or trauma of the latter may readily encroach upon the vitally important parts which surround it.

The outer muscular coat of the esophagus has an external layer of longitudinal and an internal layer of circular fibers. Near the pharynx the muscle fibers are striped, but below they are mostly of the involuntary, unstriped variety.

Between the mucosa and the muscular coat is a thick submucosa, imbedded in which throughout the lower half of the esophagus are mucous glands. The mucosa lining the esophagus is protected by dense stratified epithelium. At the cardiac extremity, reaching upwards one or more centimeters, the protective epithelium may be lacking, the mucosa having the glandular and other characteristics of the gastric mucosa.

The act of deglutition is a complicated one, voluntary so far as concerns the fauces both voluntary and involuntary in the pharynx, and involuntary in the esophagus. A bolus of food, having been forced past the cricoid into the gullet, puts the muscle fibers of the tube under tension. This tension gives rise to peristaltic waves, which carry the food onward through the cardia. Cannon, has shown that under normal conditions the vagus provides the esophagus with a proportion of its normal tonus, and that it is necessary for this tonus to exist in order that normal physiological peristalsis may occur. Under pathological conditions in which there is a deficiency of tonus, peristaltic waves may be excited as a result of tension made upon the walls of the gullet by food. Under this condition, however, deglutition is em-

<sup>&</sup>lt;sup>1</sup> Arch. of Inter. Med., Oct. 15, 1911.

barrassed and dilatation of the tube is easily produced, except when the cardia is correspondingly relaxed. Esophageal deglutition is much favored through the abundant mucus secretion in the lower part of the tube, and in a proportion of cases in which the esophagus is in a state of atony, a like condition is present in the cardia, so that swallowing is successful although delayed.

Through disturbed innervation of, or the presence of irritation at the cardia, the passage of food may encounter obstruction at that point, and the food accumulates at the lower end of the esophagus. Depending upon the tonicity of the gullet, there may result vigorous peristalsis which continues until the muscles are fatigued; or there may occur but feeble peristaltic waves, the esophagus relaxing and perhaps dilating. This principle is concerned in the general subject of obstruction.

## OBSTRUCTION OF THE ESOPHAGUS

Obstruction of the esophagus may depend upon conditions within the organ or upon disease outside of it. Of the conditions that exercise obstructive action from the outside, we should consider (1) those that make pressure upon the esophagus and (2) those that make traction upon it.

- (1) Causes of outside pressure are aneurysm of the aorta, especially of the arch; cancer; Hodgkin's disease; lymphatic enlargement from other causes; pericardial effusion; disease of the vertebrae; abscess, and, rarely, thyroid or thymus enlargement.
- (2) Instances of traction causing obstruction are almost always secondary to old inflammatory processes, adhesions to the esophagus or scar formation with contraction.

The latter condition leads in time to the development

of a diverticulum, for eventually pulsion from the act of deglutition assists the traction in diverticulum formation. (Fig. 60.) The deformity of the esophagus whether it comes from pressure or traction, but especially the



Fig. 60.—Traction Diverticulum of the Esophagus, Communicating with the Trachea.

former, may lead to erosion, infection, ulceration and spasm.

Obstruction from conditions within the esophagus may be secondary to paralysis of the vagus nerves; I have encountered it after diphtheria and with ascending paralysis.

The more frequent sources of obstruction are (a) spasm and (b) structural changes.

Spasm of the esophagus may be temporary or transient and of minor importance; it may be persistent, progressive and accompanied by dilatation of the esophagus at a point proximal to the spasmodic contracture.

The cases of what might be designated as minor spasm of the esophagus are relatively frequent. Some patients have a life history of over-sensitiveness of the gullet. That is, certain articles of food when swallowed habitually stick, causing disagreeable substernal sensations

and at times dull pain. A. L. Benedict <sup>2</sup> refers to this as an esophageal neurosis. When the stomach tube is passed it meets with delay or stoppage from clutching of the esophagus, sometimes at one point, sometimes at another. Frequently this occurs near the cricoid, most often at the cardiac end. The condition is not a serious one in itself, although it may be related to the more serious cardiospasmus.

## CARDIOSPASMUS

Pathological Physiology and Morbid Anatomy.—Our knowledge of spasm with dilation, the so-called idiopathic dilatation of the esophagus or cardiospasmus, has come mostly within the past ten years. The disease is not infrequently met with, but formerly its nature was misconceived. States that were once regarded as diverticulum. tumor or atresia are now often found to be idiopathic dilatation or dilatation secondary to cardiospasm. spasmodic contraction is peculiar. It is usually located at the terminal end of the esophagus, hence the name cardiospasm. It is often associated with a less resistant and less persistent spasm near the cricoid. There are intervals of improvement, rarely of entire relief, and there are periods of nearly complete obstruction. The explanation of these variations is not clear. In time considerable dilatation occurs above the constriction analogous to that found in stricture of the urethra, bile duct or blood vessels; to that of the heart in aortic stenosis, the bronchioles in narrowing of the bronchial tubes, or the stomach in pyloric atresia. The extent of dilatation in ratio with the degree and duration of the spasm is sometimes remarkably great. This has led to the hypothesis that the dilatation is in some cases idiopathic and not

<sup>&</sup>lt;sup>2</sup> Am. Jour. Med. Sci., Aug., 1904.

wholly secondary to the spasm. The enlargement is commonly fusiform; in case the esophageal walls are weakened on one side, sacculation may take place.

Diagnosis.—Liquids usually pass with some delay into the stomach. Solid foods stick, cause distress or pain and are regurgitated without digestion, free from traces of gastric secretion, perhaps at once, perhaps hours after ingestion. In some cases a history reaching back many years is elicited of inconvenience in swallowing eventually becoming worse, the patient suffering a feeling of pressure or pain upon eating. In others the obstruction comes on more suddenly and may follow in the train of an illness. Hunger is complained of except when the sympathetics are much perturbed, when there may be anorexia. Occasionally the heart is functionally disturbed. The imagination is excited, some patients persuading themselves that they harbor a parasite with vicious tendencies.

The physical examination shows on auscultation, delay in deglutition murmurs.

The deglutition murmurs first noted and explained by Meltzer, are classified as primary and secondary. The primary murmur is produced by the pharyngeal contraction, pressing the fluid by the cricoid; the secondary murmur is caused by the escape of fluid from the esophagus into the stomach. They differ somewhat in character; the secondary normally occurs between ten and fifteen seconds after the primary. It is recognized best by the stethoscope applied near the ensiform or laterally over the eighth intercostal space. In obstruction at the cardia this murmur may be delayed thirty seconds or more. The stomach tube meets with positive obstruction at the cardia. By careful manipulation the tube may be insinuated for an inch or more, when it is grasped by

the spasm and held. At other times the tube will not engage, but comes to an abrupt stop and bends in the widened esophagus. Not infrequently the tube meets with temporary and at times decided opposition at the cricoid which is however, soon overcome; the spasm relaxes and the tube slips down readily until stopped at the cardia. A flexible sound having graduated olive-shaped tips (Trousseau's bougie) may be passed through the constriction. Care must be taken that the instrument is in the passage before pressure is made. For the precise location of a constriction, Schreiber's sound with dilatable bulb may be used. It consists of a flexible staff carrying a rubber tube by means of which air may be conveyed to a dilatable portion near the tip. This having been passed by a narrowed place in the esophagus is then dilated and serves, when the attempt is made to withdraw it, to locate the obstruction. Bassler suggests using a rubber bulb which may be passed into the stomach, then slowly inflated and drawn back to the cardia, thus obstructing it so that a bismuth mixture may be held in the gullet, enabling one to obtain an X-ray outline of the esophagus with any irregularities that may be present.

In the use of sounds when there is obstruction of the esophagus there is danger of abrasion or puncture of the gullet. After a sound or a relatively stiff tube is passed, a larger size should be used, and finally there may be introduced a tube, through which stomach contents may be aspirated, the stomach irrigated or gavage practiced. In difficult cases the first passage of a bougie should be directed by sight, through the esophagoscope.

The esophagoscope in skilful hands enables one to see the constriction; also the possibility of growth or ulcer may be excluded. With the expert use of the fluoroscope a shadow of the esophageal widening may be obtained, after the introduction of a bismuth mixture or a radiogram may be secured, a method that is strongly to be recommended.

DIFFERENTIAL DIAGNOSIS.—The differential diagnosis should attempt to exclude ulcer, atresia resulting from contracting scar (from swallowing escharotics or mineral poisons), cancerous and granulomatous growths, external pressure upon the esophagus and diverticulum.

Stricture from scar may be recognized through the history of corrosive poisoning and subsequent events; growths or ulcer by means of the history, the cachexia, the gross and microscopic examination of the material obtained from the sac, and by the esophagoscope; external pressure by means of physical examination of the chest, the symptomatology and history of the case, the behavior of the stomach tube and by the X-ray shadow.

A diverticulum is much more rare than was formerly supposed. Its presence is best recognized by the X-ray and the fact that the tube sometimes is passed without trouble while again it is stopped suddenly; or, a small sound being lodged in the sac, a narrow tube may be passed beyond it into the stomach. Also by the use of sounds or tubes provided with a wire, we are able to obtain a radiogram that assists materially in the diagnosis.

Analysis of Cases.—I have records of a series of 52 consecutive cases including all degrees of obstruction to the passage either of the stomach tube or food, from a state of mere hesitation or delay at the cardia to that of absolute stoppage, cases of diverticulum and of pressure from causes outside the esophagus are excluded. The ensuing facts are noted.

Twenty-nine cases showed temporary, spasmodic overcontractility generally at the cardiac extremity alone, sometimes also, at the cricoid. Two cases followed the swallowing of escharotics; one from lye, one from carbolic acid. Nine cases were from cardiospasm, all but one presenting signs of greater or less accompanying dilatation. Twelve cases were from infiltration of the esophageal walls with carcinomatous or other growth. It is interesting to note that the disease appeared either at the cardia or the cricoid, usually the former alone.

Of the 29 cases of minor spasm, there were 17 males and 12 females. The age question was negligible. A great variety of associated conditions and symptoms were present: achylia gastrica, hyperchlorhydria, subacidity, hyperesthesia, peptic ulcer, regurgitation, eructation, etc.

The 9 cases of true cardiospasm were in patients between 33 and 53 years of age. It was found in 2 men and 7 women, a ratio the reverse of that found in malignancy. Spasm of the cardia alone was present in 2 cases; associated with lesser spasm in the upper esophagus in 6 cases; with gastric spasm in one.

Gastric contents was secured in 6 cases; there was normal acidity in 2, hyperacidity in 1 and hypoacidity in 3. The contents was not obtained in the remainder. In 3 there was marked dilatation of the esophagus, in 1 it was inappreciable and in 1 there was a diverticulum. There was anorexia in 2 cases and hunger in 4. Pain while eating was present in 2, after eating in 3, distress independent of meals in 1, and epigastric pain in one.

Regurgitation of esophageal contents was the rule and in one true vomiting occasionally occurred.

Gastritis was present in one case, gastroptosis in two, irritable gall-bladder and appendix in one, and chronic indurative pancreatitis in one. In only one case was there found to be a neurotic history; this was a case of neurasthenia following an attack of cerebrospinal fever.

Case No. XXXIV. Two Sudden Attacks of Cardiospasm: Interval of 18 Months Without Symptoms: Obstruction Complete: Well After Ten Days Treatment.— A man, aged 33, had a history of severe gonorrhea, which was followed by cardiospasm. He lost thirty pounds in weight in a short time. The attack gradually subsided. One and one half years later, without assignable reasons, the spasm recurred. He was in the wilds of Canada and had to travel three days to reach me. By the free local use of cocain and anesthesin I was enabled to pass first a bougie, then a small sound and finally, a small stomach tube, number 24 French scale. There was moderate spasm at the cricoid and complete stoppage at the cardia. The stomach was irrigated and a pint of raw eggs and milk introduced through the tube, the first food taken in four days. By repeating these maneuvers daily, using larger and larger tubes and by gentle stretching of the cardia, the spasm disappeared and in a fortnight the man returned to his business and has continued well. Overdistension was not practiced.

Case No. XXXV. Preceding History of Cerebrospinal Fever, Neurasthenia; Attack of Minor Cardiospasm, Subsequently Second Severe Attack; Spasm Varied in Intensity From Day to Day; Gradual Recovery.—A neurasthenic (previously alluded to) was suffering from a second attack of cardiospasm. By gaining the woman's confidence, using mental suggestion and applying local anesthetics, lavage was at length successfully employed. Thereafter the treatment was directed to the general health, which steadily improved and the patient became able to take solid foods. It was necessary for her to select her food with care, to masticate thoroughly and to eat slowly. Ultimately she completely recovered and without the practice of over-distension.

Case No. XXXVI. Symptoms of Cardiospasm, Slowly Increasing for Years; Much Associated Spasm at Cricoid; Achylia Gastrica; Dorsal and Substernal Pain; Regurgitation; Moderate Improvement.—A married woman, aged 44, without apparent reason although there was the history of hemoptysis and an irritable stomach 15 years before, slowly developed cardiospasm with occasional minor contraction in the upper esophagus. Her general health was fairly good and without history of hysteria. The condition varied in intensity from time to time. There was considerable widening

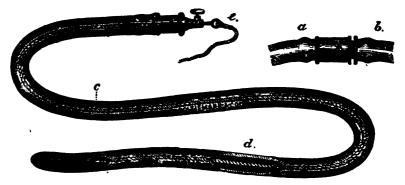


FIG. 61.—STOCKTON'S COMBINED STOMACH TUBE AND GASTRIC ELECTRODE.

of the esophagus above the cardia. Regurgitation was experienced for years and, for the previous eleven months, after every meal. As she had not become greatly emaciated it was assumed that a fair amount of food passed onward. There was complete achylia gastrica and moderate gastrectasis. Pain extending to the dorsum was experienced when lying on the back at night. Curiously enough on some days she could eat without trouble. She insisted that the drinking of cold water within two hours of eating invariably led to regurgitation. She was treated by lavage, gradual dilatation and the continuous electric current applied by means of a special electrode.

She improved, but disappeared from observation before cure.

Doubtless this case should have had wider stretching of the cardia.

Case No. XXXVII. Symptoms Appearing Gradually, Beginning Years Ago; Sticking of Food; Pain During Meals; Pain Referred to Dorsal Spine, Cure.—A married woman, aged 38 years, was overworked caring for sick ones in her family. She had had three induced abortions; nevertheless, her general health was not bad. Four years previous to examination there began a sensation of sticking of food in the gullet accompanied by a sharp, piercing pain during meals. This pain continued some time after eating and was referred to the dorsal spine, where there was tenderness. She was also tender over the gall-bladder and appendix. There was constipation, disturbed sleep and nervousness.

By means of the Sippy dilator the cardia was stretched, lavage practiced and a careful régime established. The patient was discharged as cured. One year later she wrote me that she continued practically well, but experienced suggestive symptoms when she became careless in diet or was overfatigued.

Case No. XXXVIII. Increasing Obstruction for Years; Enfeeblement Marked; Mikulicz Operation not Successful; Treatment Abandoned, Death from Exhaustion.—This case was that of a married woman, aged 53; climacteric five years previous. For 20 years she had experienced difficulty in swallowing hard or dry food. This trouble had increased of late, accompanied by regurgitation, eructation and distress or pain after taking food. At times it was impossible to swallow liquids. The patient resented local treatment and was panic-stricken. The esophagoscope revealed a tightly closed

cardia that did not relax under anesthesia. Gastrostomy was advised for temporary relief but was refused. Ultimately the Mikulicz operation (opening the stomach and forcibly dilating the cardia from below) was performed. Apparently the stretching was insufficient. The patient was not improved; gastrostomy was done to save life; other treatment was abandoned and some months later the patient died of exhaustion.

The patient refused to make the trial, but I fully believe that she might have been cured by fully stretching the cardia from above.

In consultation with Dr. McGuire I have met with two cases in which there existed at the same time spasm at both cardia and pylorus. After gastrostomy in one case it became necessary to make a gastrojejunal anastomosis. I have encountered other cases of this occurrence of double spasm which leads to the suspicion that in some instances the cardiospasm may be secondary to irritation lower down in the digestive tract and that it may be analogous to spasm of the pylorus induced by disease of the appendix or gall-bladder.

These case histories illustrate the variations observable in this affection. While some cases are transient and mild in behavior, others are sudden and threatening; still others are intractable and permanent unless treated; but in all may be seen characteristics that unite them as members of a group having a common pathology but modified by circumstances or inherited peculiarities. Enormous dilatation of the esophagus may occur in these cases. (See Radiogram XV.)

Prognosis.—When left to itself in the more favorable cases the disease has remissions followed by recurrences, any one of which may be critical. In unfavorable cases the patient becomes worn out from starvation and

suffering and dies from exhaustion or intercurrent disease. When properly treated most cases recover.

Treatment.—The first step should be the careful introduction of a bulbous tipped sound, to be followed by the passing of a stomach tube. By the application through a tube of a few drops of a 4 per cent cocain solution, or 10 gr. of anesthesin in a spoonful of water, to which should be added a few drops of adrenalin solution, we may induce a partial relaxation of the spasm. Full anesthesia is rarely necessary in order to pass an instrument: Tubes, increasing in gradation, should be passed, using lubricants freely and ending with gavage. When the spasm persists some form of dilator should be employed to stretch the cardia. I have used the dilator devised by Sippy of Chicago. Similar instruments have been devised and employed by Russell, H. S. Plummer, Jesse Myer and others. In some cases there is trouble in introducing the instrument. There is danger in over-distension, and this has led to a number of fatal results.

I have been accustomed to govern the degree of pressure by the capacity or circumference of the silk-covered bag and by the sensation of the patient, desisting when decided pain resulted. Such criteria are probably insufficient, as H. S. Plummer<sup>3</sup> who reports a remarkable series of over 90 cases, relates one instance in which rupture of the gullet was produced at a pressure recorded by the manometer at 720 mm. of mercury, yet the patient did not experience unusual discomfort. From Plummer's experience it would be inferred that a pressure of from 500 mm. to 675 mm. is indicated and practicable. For dilating the cardia, Einhorn has devised an instrument that operates on a different principle. Its char-

<sup>8</sup> Jour. Am. Med. Assoc., June 29, 1912.

(By permission of Dr. Einhorn.

acteristics will be understood by examination of the accompanying illustration. Although I have not used the instrument I can recommend it because of the results that I have seen.

By the exercise of judgment and care in dilatation the cardiac ring may be stretched so that the spasm disappears. The treatment may have to be repeated. The Mikulicz operation is formidable, and besides it may be more difficult to pass a dilator into the cardia through the incised stomach than by way of the esophagus. The danger of over-stretching is the same with either method.

It is generally admitted that the dilatation of the esophagus remains even after the stretching of the cardia has succeeded in overcoming spasm and obstruction.

Jesse Myer has demonstrated this by radiography, having employed the bismuth-containing rubber bag. Unless strongly contraindicated, an esophagoscopic examination should precede the forced stretching in all cases where the idea of a structural basis for the spasm is entertained. In case of carcinomatous infiltration or ulcer, dilatation should be discouraged.

FIG. 62.—EINHORN'S CARDIO-DILATOR: a, expanding end; aa, expanding end when giving maximum dilatation; b, flexible shaft; c, pilot wheel; d, handle and casing for actuating mechanism; e, flexible spiral shaft enclosing transmission wire; f, scale.

If driven to extremity a gastrostomy may be done for the purpose of feeding the patient. In all but one of my cases, including some not in this series, the use of the bougie and tubes and the dilating rubber bag has been successful.

There are cases of cardiospasm accompanied by extensive dilatation of the esophagus, the redundancy of which, just above the contracted cardia, leads to a pouchlike distension. Under these circumstances a sound introduced through the esophagus does not reach the cardiac orifice, but passes to one side of it into the pouch. Occasionally this difficulty may be avoided and a sound made to pass the cardia by the aid of the esophagoscope; at other times even this method has failed. It is for such cases as these that Dr. Willy Mever 4 has devised the operation of esophagoplication. In the light of his experience he recommends this method in cases in which the cardiac spasm is intractable to other methods of procedure. Following this operation for overcoming the esophageal dilatation the case may or may not require stretching of the cardia. When stretching fails to overcome the cardiospasm, cardioplasty, permanently widening the opening into the stomach, has been advised.

In mild cases much may be gained by psychotherapy, and by measures that induce sleep, rest and better nutrition. A liquid or semi-liquid diet should gradually give way to one of selected solid food most carefully masticated and slowly swallowed.

## **OBSTRUCTION DUE TO STRUCTURAL DISEASES**

These may produce complete or partial obstruction. The former most often are congenital in nature.

# CONGENITAL DEFECTS

Congenital defects include certain forms of diverticuli; \*Am. Jour. of Surg., June, 1912.

obliteration at the lower end of the tube, suggesting imperforate anus, with which in fact it has been associated; a diaphragm-like closure, often at the beginning of the lower third, with dilatation of the esophagus above, sometimes associated with a sinus communicating with the trachea or bronchus. Finally, the entire gullet may be represented by a solid cord, or the esophagus may be entirely absent.

The diseases causing partial obstruction include local infection following trauma, the exanthemata and other general infections.

# INFECTIOUS GRANULOMATA

The infectious granulomata rarely attack the esophagus. There are undoubted instances of tubercular involvement, but these are more often from extension of the disease from the pharynx and other surrounding parts. Syphilitic infiltration of the esophagus is reputed to be rare, but probably occurs more often than medical literature indicates. Not a few cases of stricture of the esophagus appear in those having syphilitic history and are greatly benefited by large doses of potassium iodid. Demonstration by autopsy of this location of syphilis is unusual. I have seen a case which, upon esophagoscopy, was decided to be carcinoma, make rapid improvement when mercury was given by intramuscular injections.

# TYPHOID ULCER

This may occur in the esophagus. Its presence is usually announced by hemorrhage, which is sometimes profuse. I met with a case of fatal hematemesis from typhoid ulcer in this region.

## PEPTIC ULCER

This may occur at the lower end of the esophagus. It is interesting to inquire how this may bappen at a point

removed from the corroding effect of the gastric juice. Probably when the peptic ulcer occurs at this point it is because the mucosa at the extreme lower end of the esophagus in such cases has the characteristics of the gastric mucosa and actually secretes hydrochloric acid and enzymes. The most typical and constant symptom of esophageal peptic ulcer is severe pain on taking food. The pain is located at the lower end of the sternum and slightly to its left, but is also felt between the shoulders. Dysphagia is present in half the cases; and early vomiting, which appears to depend upon reflex spasm, is rather constant and is an important diagnostic point between esophageal and gastric ulcer. Hematemesis occurs with relative frequency and owing to the plexus of veins in this region is sometimes most difficult to control. Perforation has occurred in a number of cases, sometimes into one pleural cavity, sometimes into the other, and even into both at once. This is accompanied by pneumothorax. At other times the perforation may be into the mediastinum, giving rise to abscess, and perhaps to interstitial emphysema or into the pericardium producing a septic pericarditis.

Treatment.—The diet should be limited to milk. Should this cause pain there should be a period of fasting, during which rectal alimentation is carried on. The coagulability of the blood should be maintained by the intermittent administration of calcium lactate. If hemorrhage occurs "coagulose" or sterile horse serum should be injected subcutaneously, or intravenously, and perfect physiological rest of the part should be enjoined.

# CICATRICIAL STRICTURE

Cicatricial stricture is most common in the region of the cricoid, but may occur at the cardia or anywhere between these points. While this may result from local ulceration, it is more often occasioned by the swallowing of escharotics. The passage through the esophagus may be obliterated or remain as a narrow and tortuous canal. From starvation, the stomach may become greatly contracted. (See Radiogram XXI.)

Diagnosis.—In case of cicatricial stricture of the esophagus there is history of a progressively increasing dysphagia usually subsequent to local injury, especially from escharotics. It is necessary to exclude functional or spasmodic cases and those dependent upon pressure from without or tumor in the walls of the gullet. The patient should be interrogated closely for true cicatricial stenosis can be traced usually to an antecedent trauma. Confirmatory evidence is to be found in auscultation and in the employment of bougies. Esophagoscopy, while the most reliable method, occasionally fails because of the spasm attending its use; but in skillful hands success is almost invariably attained.

Radiography and radioscopy are invaluable in obtaining the needed understanding of the actual condition of the gullet, at, above and below the stricture. Rules for interpreting the bismuth shadow are given by Moure, which a moderate experience leads me to confirm. They are as follows: In case of neoplasm, the pouch about the atresia is short and wide.

In case of spasmodic contraction the bismuth meal passes in jerks (à-coups) rather quickly along the dilated area without being long delayed, whereas in case of cicatricial stenosis the dilatation is long and narrow. (See Radiograms XV and XVI.)

The symptoms of spasm or stenosis at the cardiac end

<sup>&</sup>lt;sup>5</sup> Association Française de Chirurgie, XXV Congress, 1912.

<sup>&</sup>lt;sup>6</sup> Bull. Méd., Oct. 9, 1912.



Radiogram No. XIX.—CANCEROUS IN-FILTRATION AT THE PYLORUS AND GREATER CURVATURE.



Radiogram No. XX.—SCIRRHOUS IN-FILTRATION AT PYLORUS AND GREATER CURVATURE. Moderate Dilatation and Atony.



Radiogram No. XXI.



Radiogram No. XXII.

Radiogram No. XXI.—Atrophy of Stomach Following Stenosis at Cardia. (Kindness of Dr. Leonard Reu.)

Radiogram No. XXII.—FISH-HOOK TYPE SHOWING MARKED Prosis. The upper third of the stomach is occupied by a large gas bubble. The middle third, through contraction, resembles the intestine in form. The lower third, containing most of the bismuth, shows zones of distention and others of almost complete contraction. This stomach is of the fish-hook type and shows marked ptosis.



of the stomach are closely paralleled by those of "gastric sclerosis." This latter condition, usually the result of diffuse carcinoma, may also depend upon cicatricial contraction from swallowing escharotics, and in rare cases from chronic contracting perigastritis. The stomach is greatly reduced in size, the pylorus may be rigid and perhaps narrow, while the cardia remains patulous. It is under these circumstances that the stomach tube is arrested at a point that suggests cardiac obstruction; only a small amount of food can be taken before there is excited a feeling of distress, and this small meal is regurgitated precisely as in esophageal obstruction.

Treatment.—Cicatricial stenosis of the esophagus may be relieved by the practice of slow dilatation. Difficulty is often experienced with the first passage of the sound, but subsequently this decreases with each dilatation. When there is a narrow passage the introduction of an instrument is accompanied by embarrassment resembling that met with in deep urethral stricture, and is overcome by having recourse to similar methods. Often a filiform bougie must be used first. When this has been successfully passed through the stricture, a larger sound may be screwed on the proximal end, the filiform pushed onward into the stomach and thus the larger tube may be guided while being insinuated until moderate dilatation is accomplished. Preceding instrumentation, eucain, cocain, adrenalin or antipyrin may be applied locally for the purpose of relieving pain and preventing spasm and shrinking the tissues.

Several years ago a method of dilating a strictured esophagus was suggested by Mixter of Boston, consisting of passing a string of beads down the esophagus into the stomach. By drawing the end of the string through a gastrostomy opening, and by using beads gradually in-

creasing in size, it is possible to dilate the narrow passage.

Sippy has devised a successful method for dilating esophageal stricture without necessitating a gastrostomy. I quote Sippy's description of the apparatus and the method of using it.

"A foot or more of ordinary silk twist, such as Belden or Corticelli, size D, is placed in a small capsule or wadded up in a piece of chocolate candy and swallowed. After about an hour the spool is slowly unwound, so that three or four yards is swallowed during the first twenty-four hours. Subsequently from 1 to 3 yards may be swallowed each day. The taking of food and water facilitates the passage of the thread into the stomach. If the stricture is extremely tight only a small amount of water should be swallowed at one time. If the esophagus is overfilled, its contents, including the thread, is likely to be regurgitated. A small twisted silk thread will eventually go through any stricture that will permit the passage of even a small quantity of water. After the silk reaches the stomach, the normal peristalsis carries it onward. Usually at the end of twenty-four hours the thread that was first swallowed becomes deeply anchored in the intestine. It later passes out through the rectum. The thread is ready for use as soon as it is determined that it is securely anchored by pulling back on the end attached to the spool. The dilator (Fig. 63) consists of a series of graduated conical metal bulbs (A) that may be screwed on to a very flexible spiral introducer (B) 20 inches long, made of piano wire, size No. 8. Each conical bulb is provided with a central canal that is continuous with the lumen of the spiral introducer when the bulb is adjusted. This canal is large enough to glide readily over the piano wire guide (C). The guide is four

feet long and made of piano wire, size No. 20. A small perforated metal bulb, size No. 10, French scale, is firmly secured to one end of the wire by screw and solder. For

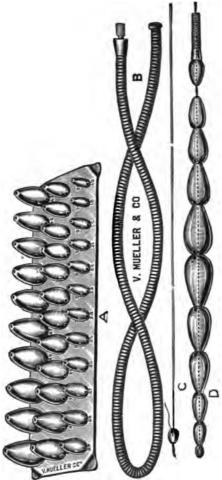


Fig. 63.—SIPPY DILATOR.
By permission of Dr. Sippy.

a distance of 8 inches adjacent to the bulb the wire is reduced in size to increase its flexibility.

"The silk thread protruding from the mouth is first drawn back from the esophagus until it is moderately

taut. The thread is then passed through the perforated bulb on the end of the piano wire guide. Holding the silk thread taut with the hand, the wire guard is introduced into the esophagus. The bulb follows the course of the thread and carries the wire safely through the stricture to the stomach. The lower end of the wire should be passed at least 4 or 5 inches beyond the lower end of the esophagus. If the thread is held firmly no harm can result even if the wire enters the pylorus. The wire is easily held in position and serves as a firm guide for the conical bulbs used in dilating the stricture.

"The diameter of the stricture is next determined by attaching conical bulbs to the spiral introducer, and passing the bulb and introducer over the wire and through the stricture. Beginning at small size bulbs, larger ones are substituted until one is found that passes through the stricture area snugly, but without the use of force. A bulb slightly larger in diameter is selected with which to accomplish the first dilation. The following procedure is advised:

"A bulb several sizes smaller than the diameter of the stricture is first threaded point downward over the wire guide. A larger bulb is made to follow this and then one yet larger. A similar cone of two or three bulbs is next threaded with points upward. The spiral introducer with a small-sized bulb attached is next threaded on the wire. The operator then holds the end of the wire guide firmly in one hand. The detached bulbs sliding on the wire are then pushed down through the stricture by the spiral introducer. The first bulb, being several sizes smaller than the diameter of the stricture, enters without friction, opening the way for the slightly larger bulb immediately behind it. The next bulb, being still larger, prepares the way for the dilating bulb, which

enters the stricture in such a way as to exert an almost purely lateral or dilating pressure. All the bulbs are pushed through the stricture and into the stomach. The bulbs are drawn back through the stricture by means of the wire guide. As the guide is withdrawn the tiny bulbous point, securely fastened at the lower end, comes in contact with the lowest conical bulb, which forces all the other bulbs backward through the stricture. The small bulb at the end of the introducer opens the way for the conical bulbs threaded with points upward. The stricture is thus gradually opened from below, so that the dilating bulb enters the stricture both from above and below with the least possible traumatism to its walls. The pressure exerted in forcing the bougie is applied in such a way as to act almost entirely as a dilating force. The operator is enabled to judge with a great degree of accuracy the readiness with which the tissue of the Thus friability of tissue, with perhaps stricture vields. increased dangers, or firm connective tissue requiring more force, may be suggested. If thought best, one or more larger sized dilating bulbs may be used in the same manner at each treatment. The rapidity with which a stricture may be safely dilated is influenced by the character of the stricture, its length, the dilatability and friability of its tissue, and such factors as pain, hemorrhage, inflammatory reaction, and other conditions peculiar to the individual case."7

The methods which under my observation have given the best results are: gradual dilatation during a number of weeks; dilatation associated with electrolysis; and, when these are not successful, gastrostomy in order to admit of satisfactory alimentation.

<sup>&</sup>lt;sup>7</sup> Sippy: "Diseases of the Esophagus," from Forchheimer: "Therapeusis of Internal Diseases."

Lerche of St. Paul <sup>8</sup> describes three cases of an unusual form of obstruction of the esophagus, consisting of a membranous growth located in the cricoid region. The diagnosis was made by the esophagoscope. The condition had existed in each patient for several years and resulted in the inability to swallow any but liquid food. There was found in each case nearly complete obstruction of the tube by a membranous band, the cause of which could not be explained. After removal of this membrane, the patient was cured in each instance.

# CANCER OF THE ESOPHAGUS

In two cases of cancer in my series of twelve there was encountered near the cricoid a marked but temporary spasm to the passage of the tube, although the tissues at this point were healthy. The tube was made to enter the stomach in two cases; in ten it failed. A bougie passed in three in which the tube was unsuccessful. In one there was sacculation of the esophagus. The youngest patient was 43, the oldest 72. There were ten men and two women in this series, which shows a susceptibility in marked contrast to cardiospasm.

Collected statistics show that three-fourths of the cases occur in males. Streaks of blood on the tube and other evidences of ulceration were present in most cases. The patient complained that passing the tube caused pain. The primary form is the squamous epithelial type, similar to that observed so commonly in the lip. It occurs isolated or in widespread infiltrations beginning in the mucosa and thence extending to adjacent tissues and organs. The mucous membrane becomes greatly thickened and its surface, rough and lobulated, projects into the lumen of the esophagus. The bronchial, tracheal and mediastinal

<sup>8</sup> Jour. Am. Med. Assoc., Apr. 19, 1913.

lymph nodes are often involved secondarily, while the cervical lymph nodes usually escape.

Diagnosis.—Early in the development of cancer as a result of local irritation there often occurs a spasmodic condition of the stomach which may be mistaken for car-



FIG. 64.—CANCER OF ESOPHAGUS.

diospasm. Any doubt may be dispelled by the use of the esophagoscope. In the more advanced cases, the radiographic shadow would assist in the diagnosis. After the passage of the stomach tube or bougie there is usually

found on it blood streaks, or blood-stained mucus, pus cells and perhaps fragments of the tissue. It is important to exclude syphilis with a negative Wassermann. The therapeutic test should be made, unless the case is unmistakable.

Meltzer pointed out the characteristics of the swallowing sound or deglutition murmur in the esophagus. He also called attention to the fact that in cardiospasm there is complete absence of swallowing sound or it may occur after very long delay, whereas in cancer of the esophagus the swallowing sound is present although differing in character from that of the normal deglutition murmur by lacking fullness and having an imperfect or broken quality of sound. The stenosis from infiltration shows in radiograms. (See Radiogram XVII.)

Treatment.—Temporary relief in early cases is obtained by the use of the stomach tube, which fact may mislead one as to the nature of the case. As constriction advances, for the relief of dysphagia, eating should be preceded by swallowing a little orthoform with a few drops of adrenalin solution. The latter also may be administered in the form of a soluble pastille.

Einhorn has found temporary relief following the use of radium, for the application of which he has contrived a special instrument.

A gastrostomy is advisable for the relief of irritation and to avoid starvation. In several of my cases the operation was accomplished with marked amelioration of symptoms and temporary gain in weight.

The surgical removal of cancer at either extremity of the esophagus, though a most formidable undertaking, has been carried out. The resection of the middle third of the gullet, which part is most often involved, owing to its relations with the aorta and the vagus nerves, has previously been regarded as beyond the resources of surgery, though recently heroic attempts at excision of various diseased portions of the esophagus have been made by Dr. Willy Meyer. In several cases he has reached the esophagus through the intrathoracic route, the operation being carried out in a cabinet arranged with a differential air pressure, higher or lower according to indications which arise. The reports of the work so far done show a marked advance in the resources of intrathoracic surgery, and lead to the hope that ultimately the removal of small, malignant growths of the esophagus may be safely accomplished. Recently the successful resection of the middle portion of the esophagus for carcinoma has been reported by Dr. Franz Torek. 10

# RUPTURE OF THE ESOPHAGUS

This infrequent lesion is of two forms: (1), traumatic rupture, and (2) spontaneous rupture.

Traumatic Rupture.—As a result of violent compression of the thorax the esophagus may be ruptured, after which substances that are swallowed or stomach contents, when vomiting occurs, may pass into the mediastinum or pleural cavities. A number of cases are reported by Sencert.<sup>11</sup> The injury in two of these resulted from the patient having been run over by a carriage. Cases of traumatic rupture are so rare and the symptoms produced are so much confused with those produced by the injuries to surrounding structures that little is known of the course and symptomatology of the lesion.

Spontaneous Rupture of the Esophagus.—This remarkable condition occurs so rarely that it is unknown to the majority of physicians. My attention was drawn to the subject by a case brought to the Buffalo General Hospital

<sup>&</sup>lt;sup>9</sup> Surg. Gyn. and Obst., Dec., 1912.

<sup>10</sup> Jour. Am. Med. Assoc., May 17, 1913.

<sup>&</sup>lt;sup>11</sup> "Des Maladies de l'Estomac and de l'Esophage," Paris, 1913.

by Dr. Irving Johnson where it was studied by Dr. Allan Jones and others.

Case No. XXXIX. Rupture of the Esophagus by Vomiting. Previous Health of the Patient, Good.—A woman, aged 42, who previously had been in good health and who was exemplary in eating and drinking, was seized with vomiting from unknown cause. Suddenly she felt agonizing pain under the left breast and declared that she had ruptured her gullet. There was evidence of shock with marked acceleration of pulse and frequent shallow respirations. Soon afterwards the temperature was found to be 104 F., and her face and neck showed subcutaneous emphysema. The following day the pain was intense, respiration more difficult and the emphysema increased. Physical examination showed limited motion of the left chest and absence of vocal fremitus. breathing sounds and vocal resonance. The abdomen was somewhat distended and tense, but the stomach was found to be in position. There was belching of gas which had a strong fecal odor, but the vomiting ceased. To exclude possible diaphragmatic hernia or ulcer perforating the diaphragm an exploratory celiotomy was made, but nothing was found wrong in the abdomen. The patient died about forty-eight hours after the onset of the symptoms.

At autopsy the left chest was found distended with gas and semipurulent fluid, having a foul and fecal odor. The left lung was collapsed, and the pleura was covered with thick, fresh, fibrinous exudate. The tissues of the mediastinum were emphysematous. The pericardium was injected and contained a little seropurulent exudate. On the left posterior wall of the esophagus just above the diaphragm was found a vertical rent about an inch in length communicating with the posterior media-

stinum and the left pleural cavity. When the pleural cavity was incised gas escaped under positive pressure, yet no injury of the pulmonary tissue was discovered.

The history of this case is important and its careful study casts light upon the nature of some of other reported cases, the nature of which has been questioned. In 1877 Reginald Fitz 12 reported a case of spontaneous rupture of the esophagus which he examined at autopsy, the history of the case being given by the attending physician, Dr. George O. Allen. In this case there was supposed to have occurred a primary rupture which was gradually increased through efforts in vomiting. Quite unlike other cases this patient survived eight days. the great majority of cases death occurs within fortyeight hours. In reporting this case Fitz gave critical analysis of the reported cases which he had been able to collect up to date, and the diagnosis in some of them he seriously questioned. It seems probable with the knowledge obtained from the Buffalo General Hospital case that certain of these cases, the diagnosis of which was doubted by Fitz, were actually cases of spontaneous rupture of the esophagus. It should be emphasized that in the Buffalo case the interstitial emphysema did not depend upon a lesion of the lung. It also should be noted that there was no evidence of post-mortem decomposition of esophageal tissue and as the autopsy was made soon after death, there was no autodigestion. It should be noted that the patient was not a large eater and did not drink alcohol. Most of the reported cases have occurred in alcoholics and in the case reported by Fitz, delirium tremens developed soon after the rupture.

The first known case was reported by Boerhaav in 1724. From its history I assume that it should be

<sup>12</sup> Am. Jour. Med. Sci., Jan., 1877.

classed with the genuine cases. The same may be said of the case reported by Dryden in 1788. Other recent cases are described by Sencert in the work previously mentioned. In the additional case here reported the histological examination gave no evidence of previous softening, ulceration or other disease of the esophagus. It was noted that the epithelium at the lower end of the esophagus showed the glandular structure characteristic of the fundus of the stomach, and extended upwards into the esophagus in arrow-like projections, the squamous epithelium extending down between these projections, forming a sagittal union.

The epithelium adjoining the rent appeared to be healthy and apart from the superficial changes at the edges of the rent, the result of infection, the muscular layer was not different from that in other portions of the esophagus. Evidently the walls of the esophagus, although without disease and apparently normally developed, were lacking in resistive power and were torn by violence no greater than that which ordinarily attends hard vomiting.

Symptoms mark the history of esophageal rupture. There is severe pain in the region of the sternum and left breast. There is evidence of shock, soon followed by shallow breathing, accelerated pulse, cold and sweating surface of the body, elevation of temperature, leucocytosis and the physical signs of pleurisy and pneumothorax. A conspicuous symptom is the appearance of interstitial emphysema, first about the neck and face and in some cases extending practically over the entire body. In the case here reported there were eructations of gas having a fecal odor corresponding with the odor present in the gaseous and semi-liquid

contents of the left pleural cavity as found post mortem. The above collections of symptoms would seem to be sufficient on which to base a diagnosis of spontaneous rupture of the esophagus. The fecal odor of the eructations I have not found noted in the history of other cases; its significance in this case is doubtful.

Prognosis.—All cases so far reported have terminated fatally, usually before the lapse of 48 hours. The case reported by Fitz survived eight days, but that was supposed to depend upon the fact that in the beginning the rupture was small, and subsequently became larger.

Treatment.—Hitherto no treatment has been attempted, excepting measures for the relief of suffering. In view of recent work in thoracic surgery, the indications are plain that the condition should be attacked by a thoracotomy, thus exposing the mediastinum and enabling the surgeon to reach and repair the esophagus and drain the pleural cavity. So far no trial of this procedure has been reported.

## DIVERTICULUM OF THE ESOPHAGUS

This rare condition, as already stated, may be divided into traction, pulsion and traction-pulsion forms. The deformity may occur in the pharynx above the esophagus as well as at any point in the gullet.

**Diagnosis.**—The diagnosis may be made by the radiogram, the esophagoscope, or by sounding according to the method of H. S. Plummer.<sup>13</sup> (See Radiogram XVIII.)

Treatment.—The condition may be relieved by feeding regularly through the stomach tube so as to avoid pulsion with its resulting increase in the volume of the sac. Cure becomes possible only through excision.

<sup>&</sup>lt;sup>18</sup> Collected Papers, Mayo Clinic, 1905-9.

## **ESOPHAGITIS**

The esophagus is so well protected by its thick mucosa, and by its resisting deeper tunics, and so well drained and swept clean through the performance of its normal function, that infection and inflammation of the structures are rare as compared with the mouth and pharynx adjoining one extremity and the stomach and intestine at the other.

The natural resistance to injury and infection ceases to be effective when from any cause stagnation of ingesta occurs.

Infection may extend downwards from the pharynx or upward from the stomach or may result from inflammation of the mediastinum.

Infection carried through the lymphatics or, exceptionally, through the blood stream in case of general septicemia, may lead to inflammation in the walls of the esophagus or in the adjoining tissues.

Esophagitis and periesophagitis, although they may assume various degrees of intensity and persistency, are for convenience in description divided into acute and chronic forms.

## **ACUTE ESOPHAGITIS**

When from any cause there is a loss of the protective epithelium, an inflammation may be excited from the entrance into the mucosa of the ordinary saprophytic bacteria conveyed with the saliva or food. The specific infections may invade the mucosa or the deeper structures of the tube independent of preceding injury to the protecting epithelium. For instance, in diphtheria, small-pox or typhoid fever the specific bacteria or their toxins, conveyed through the lymph stream or the blood, may give rise to serious esophagitis.

Acute esophagitis may be excited also by the swallowing of scalding liquids, mineral acids or caustics of any kind in solutions of sufficient strength to cause injury to the mucosa, yet without destroying it. It may also follow local trauma to the mucosa such as comes from swallowing foreign bodies, or from too much violence in the passage of sounds or other instruments. Any of these events favor infection. In consecutive, acute esophagitis exceptionally a phlegmonous inflammation may develop with suppuration and sloughing of large masses of the mucosa. A specimen in the museum of the University of Buffalo represents the entire mucosa of the esophagus which was vomited by a patient who had phlegmonous inflammation of the esophagus induced by the swallowing of some unknown poisonous substance.

A diffuse purulent esophagitis, with the formation of abscesses, the discharge of pus and subsequent deformity from scar formation, is a rare condition which results from infection with pyogenic bacteria, which more often reach the esophagus through the circulating blood, but exceptionally are due to local infection.

Symptoms and Clinical History.—The course of a light type of esophagitis is accompanied by a sensation of burning, dysphagia and sometimes pain of a sharp character. Attempts at swallowing often induce spasmodic contraction and regurgitation of the food. In the more serious form the same symptoms appear, but are more intense and more prolonged. In cases of phlegmonous inflammation, or in those of suppurative esophagitis, the local symptoms of pain, dysphagia and spasm are accompanied by systemic disturbances, including elevation of temperature and cardiac excitement. At times through disturbance of the pneumogastric, the respirations become frequent, the patient shows depression and

shock and suffers pain upon deep breathing, cough or other movements of the chest. The least attempt at deglutition gives rise to intense retrosternal pain. Subsequent to such an esophagitis stenosis is to be expected.

Treatment.—It is inadvisable to pass sounds or stomach tubes through an inflamed esophagus. It is sometimes practicable to administer small quantities of some soothing antiseptic lotion, such as boroglyceride in one per cent solution. In other cases, especially following a burn, even the most soothing preparations are likely to increase the symptoms probably because of the movements of the esophagus during deglutition. The patient should be nourished per rectum. When there is severe pain, prostration or shock, codein or morphin should be injected.

# CHRONIC ESOPHAGITIS

Simple esophagitis may terminate in chronic inflammation. Chronic esophagitis also may be occasioned by the habitual drinking of undiluted spirits or by the frequent taking of stimulating condiments. The condition also occasionally develops in the course of tuberculosis, syphilis, diabetes or other disease in which the resisting power is low. The lesions are usually superficial, although ulceration and suppuration may result from a fresh infection of the already diseased mucosa; in other words, an acute esophagitis may begin on the surface already involved in chronic inflammation.

Bleeding from the esophagus, usually slight in amount, is not uncommon in chronic cases. On the passage of the stomach tube a large amount of mucus, often bloodstreaked, and sometimes free blood in considerable quantity, is regurgitated. When even comparatively super-

ficial ulceration occurs at the lower end of the esophagus where there is a large venous supply and where the veins are often dilated, there may be hemorrhage of a serious nature. In some chronic cases false membranes develop resembling those of diphtheria.

The symptoms of chronic esophagitis are of varying degrees of intensity; the most important are dysphagia and pain. There is anorexia doubtless from the discomfort or distress occasioned by swallowing. In mild cases the patient may take unirritating liquid or semiliquid food without serious difficulty. In the more serious types of the disease there is emaciation from the lack of food. The lowered nutrition, the continued infection and the nervous depression natural to the state, unite to produce the symptom-complex of a formidable character.

Treatment.—In simple chronic esophagitis recovery ordinarily follows when the patient forbears from taking alcohol and other irritating substances. Improvement is favored by the taking of bismuth emulsion or solution of boroglycerid. An occasional teaspoonful of a 1-500 solution of salicylic acid should be swallowed. In more serious types of esophagitis, when there is ulceration and swelling persistent in character, it is necessary to raise the patient's resisting power to the utmost. The advisability of making a gastrostomy, so that the esophagus may have rest and so that the patient may be well nourished, should be considered. The subsequent treatment becomes that for relief of esophageal obstruction.

Soothing demulcent drinks, the administration of the milk of bismuth, or milk of magnesia, or the oil of sweet almonds, in which is dissolved a little menthol or thymol, are measures which give comfort and promote recov-

ery. The diet should at first be limited to fluids, especially milk and the gruels. Later unstimulating pultaceous food should be given and followed by a weak solution of one of the essential oils or some other cleansing and non-toxic antiseptic draught. Allan Jones recommends the following prescription.

Ŗ	Bismuthi subnitratis	, ,
	Mucilaginis tragacanthae	
	Aquae menthae piperitaeaa qs. ad	120.00 e.c.—(3iv)

Sig.—Teaspoonful undiluted as often as needed to relieve burning.

# CORROSIVE DESTRUCTION OF MUCOSA OF THE ESOPHAGUS

In the museum of the University of Buffalo is the specimen of a remarkable case in which a complete cast of the esophagus and the upper portion of the stomach was vomited. Profuse hemorrhage followed and the patient died.

This destruction of the mucosa followed the swallowing of an escharotic, the precise nature of which was unknown.

## PARALYSIS AND FUNCTIONAL DISEASE

Although paralysis of the esophagus may follow central nervous disease located in the cranium or the spinal canal and may result from neuritis produced by diphtheria, ascending paralysis, and other causes, it is also true that it may be purely hysterical. Although paralysis of the esophagus is comparatively rare, anesthesia is relatively common. Another frequent disturbance is hyperesthesia, in which swallowing becomes distressing and there is morbid fear and anorexia. The condition may be associated with temporary spasm.

Treatment.-Relief of the sensory disturbances of the

gullet may follow the use of the stomach tube or the galvanic current applied with a suitable electrode. Much more important is constitutional and psychic treatment. Of the many methods of the former, the cold spinal douche, needle bath, massage and spondalotherapy are useful together with the administration of nervines, especially the mistura asafetida in full doses 15 c.c. (3ss.) twice daily.

## FOREIGN BODIES IN THE ESOPHAGUS

The presence of a foreign body in the esophagus is sometimes a misapprehension based upon the supposed swallowing during sleep of, for example, a false tooth or a coin. Often enough the presence of a foreign body in the esophagus is a reality, and one that occasions suffering or danger to the patient and perplexity to the physician. Comparatively small and smooth objects may sometimes be safely pushed into the stomach by means of a sound. But such a maneuver is unsafe when there are sharp points or edges. The removal of foreign bodies may be safely affected by an expert after examination through the esophagoscope; but rarely the dangerous operation of an external esophagotomy is necessary. Occasionally a patient complains of unusual sensations in the gullet suggesting the presence of a living and moving body. Stengel has reported cases of this sort, which had as a cause the presence of polypoid growths and these, by changing position, gave rise to the sensations complained of. As a rule these sensations are based upon hysteria or a distinct obsession, in which case great benefit usually follows an examination with the esophagoscope whereby the patient may be convinced of his false notion and by suggestion relieved of discomfort.

### HEMORRHAGE

The most frequent cause of bleeding from the esophagus is chronic congestion from obstruction, at times leading to dilatation or even to varicosis of the esophageal veins. (Fig. 65.) Such vascular distension is most com-



FIG. 65.—DILATATION OF ESOPHAGEAL VEINS. (Museum, University of Buffalo.)

mon at the lower end of the gullet where the prominent varices suggest hemorrhoids. Venous obstruction occasioning distension of the esophageal vessels and predisposing to hemorrhage, is usually secondary to hepatic disease or to portal stasis from any cause. The bleeding

is often started by slight trauma from swallowing boluses of coarse food. Once hemorrhage begins it tends to persist as a result of the unusual venous blood pressure. When bleeding of the esophagus is associated with portal stasis it may be controlled with less difficulty.

Treatment.—It is often judicious to begin with the administration of a large dose of calomel, followed by magnesium sulphate. Thereafter, styptol, antipyrin and adrenalin may be given in frequent small doses. The patient should abstain from eating and should receive rectal alimentation. The frequent swallowing of a teaspoonful of gelatin solution is of some value. When the coagulability of the blood is insufficient, calcium lactate should be given, and serum, "Coagulose," injected subcutaneously.

# CHAPTER XXXIV

#### ALIMENTATION

In order that there may be full development and that the physiological balance of good health may be maintained, it is necessary to ingest vegetable and animal food in sufficient amount but not in excess. The quantity of a meal should be suited to the digestive capacity as well as to the alimentary needs of the individual.

Intelligence must be displayed in the selection and preparation of food and too great authority to the instinct is not to be permitted in the manner of eating. The morsels should be of proper size and should be carefully masticated and ensalivated.

Economic Importance of Proper Alimentation.—Nourished in a rational manner and having a sound digestive apparatus, the individual perfects his growth, supplies himself with abundant energy and deposits in the economy a reserve for use on occasions of accidental privation or excessive demand.

With a suitable diet, with normal secretion and motion in the alimentary canal, the soluble ingesta is almost completely liquified. The available portions are appropriated and the residual waste contains a minimum of material to favor bacterial growth; but slight fermentation or putrefaction occurs, and the dejecta have a molded form, a homogeneous appearance, with scarcely distinguishable remnants of the food, and are discharged at definite intervals, habitually and comfortably.

Enjoying thus the satisfaction of physiologic needs rationally controlled, the individual attains his full de-

velopment, either avoids, or more successfully resists the accidental infections or trauma and reaches the full limit of his years. This wise course of alimentation, however, is rarely taught, enforced or practiced. Beginning in infancy, nursing pathologic mammae or unhygienic bottles; surviving an unhappy childhood, painfully sustained by innutritious, excessively rich or irritating food; passing through adolescence when fancy is active, when desires become dominant because not guided by example, reason and thoughtful instruction, man forms vicious habits. These create artificial needs and blunt the sensitiveness or obscure the dictates of normal instinct; faulty maturity is reached with a heritage and an acquired disposition for intemperate and injurious alimentation.

In such a case the individual lacks not only in development, but in power of internal resistance and in energy. His nutritive functions are in disharmony; he is unprepared for the exigencies of life; mentally, morally and physically he is below native efficiency.

The diet of an individual is often governed by the practice of family or community, by advertisements of trade, by his own vicious taste; and with abnormal psychic states, introspection and misinterpreted experience, he falls a victim to misguided prejudices.

Ethnologists account for the peculiarities of races and people by the character of their nourishment. Animal breeders and plant growers have learned that by the nutrition provided an organism its characteristics are profoundly modified, especially when practiced through several generations. Man fails to profit, so far as concerns his personal welfare and best development, by the intelligence with which he is endowed.

Although some of the most flagrant mistakes in nutri-

tion originate in deliberate intent, yet these usually spring from the teachings of opinionated "cranks," zealous mystics and impudent apostles of diet and hygiene in whom knowledge of physiology is replaced by effrontery.

An unphysiological diet may be excused when no other is available, but an emphatic protest should be sounded against those who eat unphysiologically in accordance with an unsound principle and who teach others to follow their example. Just as asceticism and gluttony are irrational, so, too, a vigorous vegetarian diet, excessive indulgence in animal food, prolonged fasts or restriction to uncooked foods are to be shunned. A rational diet, so necessary to the individual and of corresponding importance to a nation, is less often observed than is com-The proper feeding of animals because monly believed. it touches the question of economics is considered of such importance that it has interested a department of the government. The proper alimentation of the people because it touches the question of human rights of prejudice and of commercial greed, is left without guidance or is controlled by tradition, caprice, fanaticism or exploitation.

To attempt to procure the general adoption of a rational diet would be a mighty undertaking, but even a correct understanding of the problem involved would be a great help. The subject may be considered from three different angles.

- (1) A dietary based upon truly scientific laboratory examination of the ingesta and egesta and thus upon the requirements of the individual.
- (2) A dietary based upon the demands of unperverted instincts, the conscious needs and the empiric knowledge gained from experience and intelligent trial.
  - (3) A diet based upon a reasonable compromise be-

tween the first and second; one wherein the teaching of the laboratory may be humanized by regard for the appetite, and wherein the call of the instincts may be corrected by data obtained in the laboratory.

A discussion of these three points of view becomes the charge of the practitioner who has to instruct an opinionated clientèle. A knowledge of dietetics is invaluable to one who would prevent and relieve disease, especially disease of the digestive apparatus.

True, the exact methods of the laboratory, provide the means of knowing the number and character of food units which an individual needs to sustain him in growth and in a state of nutritive balance according to his rest or activity; but an estimation of the metabolic power of an individual involves expertness and consumes so much time that its practice is necessarily neglected save in the more important cases. Methods of examination need to be simplified and improved, for it is desirable to know the functional power of the liver, pancreas and kidneys as a comparatively routine practice. The examination of gastric and intestinal contents is not enough. Disturbed digestion depends not alone upon the quality of food and upon the local condition of the digestive organs but also upon the metabolism.

Over-fatigue depresses the metabolic power. By over-fatigue is meant the result of exercise of mind or body beyond beneficial physiologic limitations for the particular organism under existing conditions. Suitable exercise ordinarily stimulates the metabolism; over-fatigue weakens metabolism and prevents the assimilation of nutriment which is needed and without which there will follow loss of functional activity and weight. Disease also, sooner or later, depresses metabolic power. It should be sought to ascertain what amount of exer-

cise may be beneficially taken considering the nature, severity and duration of the disease without producing fatigue and without depressing the metabolic power. Stated differently, we should estimate the number of calories that a given patient can assimilate, considering his condition, which means his disease and his activity.

Two important factors must be weighed in coming to this conclusion. (1) We should judge the amount of energy that is expended in involuntary use of mind and body; in pain, anxiety, insomnia, delirium; in cough, restlessness, going to the toilet, etc. (2) We should consider the character of the food, its quality, the method of its preparation, to the end that the appetite may be awakened or, in other cases, left as much as possible undisturbed. Finally the digestibility of the food must be counted with due allowance for the crippled stomach.

It will be seen that repletion from the standpoint of the metabolism may be possible while denutrition from the standpoint of the general economy is going on.

The general economy may require and be ready to receive nutriment while the metabolism is unable to deliver it; the metabolism may be prepared to perform its work while the primary digestion is unable to accommodate it; finally, the primary digestion, even though diseased, may yet succeed in the necessary hydration of judiciously selected foods, scientifically and appetizingly prepared, and yet fail to digest the food that is commonly provided.

The organism, the metabolism and the digestion all fail under continued starvation; they also fail under continued over-alimentation.

A low diet, in and of itself, may be bad for weak digestion and yet be temporarily demanded for the relief of a special condition. Many cases of dyspepsia continue because the patients fail to eat enough. Also, dyspepsia is induced when the metabolism is over-taxed from superalimentation. There must exist a physiologic harmony between all these factors.

In practical dealing with cases there is need of the study of caloric values in the attempt to obtain the best diet possible for the patient, in view of all the factors involved and the relation existing between them.

A healthy adult requires in 24 hours from 14 to 20 calories per pound weight, according to his activity. A man resting in bed may require under 2,000 calories; exercising moderately, about 2,500; doing heavy work about 3,000.

This ration, as will be seen, may need to be changed in disease, and especially disease of the stomach. However, there must be kept in sight the normal standard, and the attempt must be made to reach this standard, not permitting a patient suffering from a gastric disorder to turn unaided to his deranged appetite as a guide for nourishment.

Selection of Dietary.—When digestion, metabolism and elimination are satisfactory the estimation and distribution of the number of calories composing the best diet in a given case become fairly easy.

A convenient way to formulate a diet in which the caloric values are distributed with regard to the proper amount respectively of proteids, fats and carbohydrates, is to prescribe the needed number of "portions" of the foods selected. These portions each contain approximately a given number of calories, so that it is a simple matter to indicate a meal that meets the requirements.

Useful tables representing the proportion of proteids, fats and carbohydrates present in each "portion" have been arranged by Horace D. Arnold of Boston, by A. L.

Benedict of Buffalo 1 and by others. In dealing with gastric diseases the conditions are such that often for the time being little attention can be given to caloric standards; the question is one of tolerance of food, even of the necessity of abstinence. Eventually, however, the quantity has to be considered and it is important that this phase of the question be not put aside. The timidity and fancy of the patient often tend towards insufficient eating, many times to his disadvantage. A low diet long continued weakens the digestion as it does the other physiologic activities. A patient may need to eat more food and a greater variety of food in order to strengthen his stomach just as he needs to increase exercise to strengthen his muscles.

Most dyspeptics are possessed of exaggerated fear of certain articles of food from which they have at some time suffered. Such apprehension may be well grounded as when depending upon personal peculiarity or upon food anaphylaxis; yet in most cases it is but the result of fancy.

At other times an opposite tendency has to be combated; the patient may indulge himself improperly, eat inordinately, possibly practicing deception as to his excess.

Although over-indulgence is more common concerning the use of tea, coffee, alcohol or tobacco, it is at times found in the matter of variety and quantity of food; or the manner and time of eating may be at fault. When a patient should eat and when he should fast, although important, is valuable information that may elude us. It is a subject worthy of thoughtful consideration. A day of abstinence, except for water freely given, may cut short a beginning gastritis; yet this course may increase

<sup>&</sup>lt;sup>1</sup> "Golden Rules of Dietetics."

the distress occasioned by hyperchlorhydria. The taking of condiments is likely to cause discomfort in hyperchlorhydria, ulcer, cancer or gastritis, yet may distinctly benefit the patient with atony. Condiments and other foods locally stimulating are therefore not without place in dyspepsia, although in most instances they occasion symptoms.

The question as to fluid or solid food is far from one of indifference. When practicable a meal should consist largely of solids which need to be masticated thoroughly: vet at times all solid foods should be excluded. To understand this it is only necessary to recall the mechanical side of gastric digestion. For some time after ingestion the bulk of a meal should rest in the fundus while carbohydrate digestion proceeds. This is not easily accomplished when only liquid food is taken and there are other obvious objections to a diet limited to fluids; on the other hand, the fact that fluid and semifluid foods require little peristaltic action of the stomach is a good reason for their use in numerous cases in which gastric motility is proportionately too active or in which the mucosa is oversensitive, as in gastritis or in certain cases of ulcer.

When there is marked general or local depression and therefore insufficient energy to produce active gastric peristalsis, a fluid or semi-fluid diet is preferable, at times almost indispensable. A milk diet is often the best compromise between the fluid and solid. If there is normal secretion, the milk is changed into soft curds which are retained temporarily in the fundus of the stomach, while proteolysis goes on undisturbed in the lower strait. This coagulation of milk requiring for its comfortable digestion a certain degree of motor activity, renders it unsuitable in other cases when there is marked over-

sensitiveness. Under such circumstances the milk should be peptonized or given in the form of gruel or thin porridge. At other times it agrees best when fermented in the form of koumiss, matzoon or vito-lac.

A milk diet may fail to succeed because of the manner of carrying it out. Often milk will disagree when given with other food and yet cause no disturbance given by itself and at proper intervals. The intervals and the quantity of each portion are of considerable importance.

The ordinary portion, a glassful, about 250 c.c., given every second hour may cause distress and yet this amount may be well tolerated if given every two and a half hours. Again, it may be best given at shorter intervals, and this is practicable when there is active gastric motility.

Milk should not be considered as a drink, but as a food, and there are objections to using it merely to quench thirst. When the digestion is feeble, milk should not form part of the general meal, especially when meat is taken. In case of very active gastric secretion, milk may be advantageously given with carbohydrates or with eggs. It usually requires a fairly good digestion to dispose of a conventional "egg nog" without distress. When taken very hot, yet not scalded, milk exerts a stimulating action not occurring when taken cold. Many of the apparent objections to milk result from its having been contaminated. Only recently has it become possible to obtain milk that is pure, unchanged and low in bacterial count. The slight difference in cost, however, often influences the consumer to use milk that is unfit.

Aside from danger in the bacterial content of poor milk, its occasional toxic qualities and the difficulty often attending its digestion render it unsuitable for the dyspeptic.

Animals and meat should undergo much more rigid inspection than is now practiced. Particularly is this true of meat used by the dyspeptic. There are instances in which meat is digested best when uncooked. It should then be scraped with a sharp knife, the fat, tendon and cartilage carefully removed. Thus prepared it may be served as a sandwich or may be made into small patties and slightly grilled, with a little butter spread over when served. Roasted meats are often unsuitable to an irritable stomach, first because of the unequal cooking in different parts of a roast; second, because the fats are broken up by the heat into fatty acids that, although permeating the meat and giving it an inviting flavor, nevertheless are at times highly disturbing to the digestion. Meats roasted before an open fire, as was the ancient custom, are more digestible than when ovened. Grilled meats are far preferable to those that are fried; the latter should be discarded in stomach troubles. Boiling, long continued over a slow fire, renders meat suitable for easy digestion. Gravy, meat sauces and soup stock are rarely to be admitted in the dietary of the dyspeptic, except when there is inactive secretion from functional causes. In such conditions, bouillon, broth and meat extracts are excellent stimulants to secretion and are otherwise advisable.

Beef and mutton are to be selected in preference to pork; however, pork tenderloin and boiled ham agree well with some cases; chicken is more easily digested than turkey. Duck and goose are not suited to the dyspeptic. Young chickens, the so-called "broilers," and fowls tough because of age, are to be discarded. Chicken one year old, drawn as soon as killed and not too long

hung, should be selected. Such fowls prepared by boiling or stewing are more easily digested than when roasted. Pigeon when properly prepared is more digestible than squab, though the latter is more digestible than a "broiler" chicken. Of the wild birds, snipe, woodcock and quail are preferable to partridge and other large birds. As a general rule game should be avoided in the diet of dyspeptics.

There is great difference in the digestibility of fish which depends in part upon the variety and in part upon condition and mode of preparation. Because of fat, of hardness of fiber, or special objectionable qualities, certain fish should not be included in the diet; among these are sturgeon, salmon, mackerel, sword fish, cat fish and eels. On the other hand, pike, perch, white fish, brook trout, cod, flounder, sole, halibut and weak fish may be digested more easily than meat.

Much depends upon the condition of fish in reference to the season, the water in which they are caught, the place where they are stored and, above all, their freshness, which is indispensable. Unless satisfied as to excellence of quality, we should prohibit the use of fish. In their preparation, boiling is altogether preferable to roasting or frying. Some varieties of fish when grilled, though not more digestible, are more appetizing than when otherwise prepared. Oysters fresh from the shell are usually good for the dyspeptic except when there is motor insufficiency; in that case they are very objectionable. Cooked oysters, more often than raw, cause distress and should be avoided save when there is active digestion. There is more often an idiosyncratic objection to shell fish than to other viands; however, this is less frequently true of oysters than of others in this class. The round hard-shell clams or "quahogs" are

more acceptable than the soft-shell variety. In the diet of invalids clams are rarely used except in making broth; clam broth often acts as an appetizing and nutritious gastric stimulant.

As a rule eggs are more easily digested raw than cooked. Usually they may be given successfully when partly and slowly coagulated by immersing them in a bowl of boiling water and letting them stand for eight to ten minutes.

Poached eggs, as usually served, have the white overdone and nearly hard, and are ill suited to the sick except when there is active digestion. An excellent way to serve an egg when the gastric secretion is low, is to stir it briskly into a cup of hot broth or bouillon, not allowing it to coagulate.

Another good way of serving an egg is with hot milk (lait de poule). Prepare as follows: Beat well in a bowl the yolks of two or three fresh eggs with a table-spoonful of sugar. Pour this mixture slowly into three cupfuls of milk which has just come to a boiling point.

The yolk of eggs is better tolerated by some than the white; with others the white agrees best. At times when the stomach rejects eggs otherwise prepared, the white of eggs mixed with fresh pineapple juice is retained.

In case of anacidity it is well to serve the white of eggs dissolved in a little cold water to which has been added a little dilute hydrochloric acid.

Eggs are invaluable in the feeding of certain cases when the stomach is irritable and when it is important to provide a diet rich in proteid.

With some patients, owing to an idiosyncrasy, eggs are especially toxic; with others they may be taken, but only one or two a day; beyond this number they cause

toxic symptoms, occasionally, also, gastritis. On the other hand, there are patients who are able without resulting symptoms to ingest from twelve to eighteen eggs daily.

When feeding eggs to a dyspeptic we should select those coming from hens that have been fed on proper food. The eggs coming from barnyard fowls have a disagreeable flavor, even when strictly fresh. For the sick it is well worth the trouble to secure eggs not over two days old.

A fresh egg may be recognized by the diaphanous and pinkish transparency of the shell. The older the egg the more opaque is the shell, which little by little becomes spotted in appearance upon transillumination. When a fresh egg is broken on a plate the yolk will remain in a firm compact mass in the center of the white. It will not spread or flatten.

Vegetables are usually largely excluded from the regimen of dyspeptics, partly because as ordinarily provided they are an uncertain element in diet. Much depends upon the variety of vegetables, more upon freshness, most of all upon the mode of preparation.

In gastritis, cancer and all conditions producing great irritability of the stomach, vegetables are best excluded. When local stimulation is tolerated, they may be given advantageously. At times when animal proteids are harmful because of metabolic defects, it is highly desirable to give vegetables in order to relieve monotony of diet. Although this may not be done in certain cases, still with consummate care in selecting vegetables and in cooking them, they become admissible in a wider range of cases than is commonly supposed. There is great need of reform in the supply of vegetables. As commonly displayed in markets they are under-matured, over-ma-

tured, or deteriorated by age. Commonly they have undergone fermentative and other subtle changes which make them sources of irritation, whereas originally they might have been wholesome. This most often happens with the legumes and other green vegetables, but is not without bearing as regards the tubers and roots.

An exhibition of culinary skill is particularly necessary in the preparation of vegetables if they are to be nourishing and undisturbing to the dyspeptic. As commonly served they have lost in nutritive value and are frequent causes of gastric distress.

What has been said of the quality of vegetables is true of fruits; they are often harmful because gathered when under-ripe or they are stale and undergoing fermentation. Fruits are to be prohibited in cases of gastritis, ulcer, cancer, hyperchlorhydria, ischochymia and hyperesthesia. Juice from selected oranges, pine-apples or grapes is admissible in cases of functional hypochlorhydria and, in small amounts, in gastric atony: Cooked fruits whether fresh or dried may be taken with less reservation than appertains to fresh fruits. When, from experience in a given case, the taking of fruit, fresh or cooked, or of fruit juice, is found not to disturb a patient, their use should not be prohibited.

The attempt should always be made to allow a varied diet and to avoid proscription of the foods craved and ordinarily selected by the individual. Not only may the digestive organs lose power or fail to recuperate through too great dietetic restriction, but the patient may easily fall into wrong habits of eating resulting in loss of confidence.

It is by this chain of circumstances that there is engendered habitual low nutrition with consequent lack of energy. Bread properly enters into the diet of most dyspepties. Sometimes, for reasons not clearly understood, bread made from one kind of grain is disturbing to a patient who may take another sort of bread without discomfort. As a rule, bread made from fine wheat flour should be chosen, but occasionally, even in irritable stomachs, that from whole wheat flour or graham agrees better. Occasionally bread made from rye or rye and wheat mixed proves to be more suitable than that made from the finest wheat flour.

In the northern states corn bread is usually supposed to be unsuitable for a dyspeptic, yet in the southern states, where corn bread more generally enters into the dietary of the people, it is often selected by the dyspeptic. Probably custom and education fully as much as digestibility enter into reasons for the selection by the dyspeptic of one kind of bread in preferment to another. Some persons seem to digest best hard tack, very hard biscuit or the hard bread imported from various countries. Whatever may be the variety of flour from which it is made, the bread should not be eaten until it is at least twenty-four hours old. An exception to this, is certain forms of corn bread which seem to be most digestible when fresh.

The degree of staleness before bread is suited for a feeble digestion, is to be determined by testing the readiness with which it is separated by mastication into a pap-like consistency. To be easily digested by the dyspeptic, bread should enter the stomach, not in sticky, rather tenacious lumps, but so chewed and ensalivated that it has become something resembling a gruel. It will be found that as bread is made by some bakers, this preliminary mouth digestion is not easily procured, even when the bread is several days old. Much therefore de-

pends upon how it is made; and before deciding that bread must be excluded from a diet, it is advised that its quality be carefully examined. Often all that is needed is a change from bad to good bread; that is, from underdone to well-ovened bread; from that baked in an overheated oven, giving a blackened crust and a doughy crumb, to that baked in the right temperature for a sufficient length of time.

The character of the yeast employed is a matter of consequence. Depending upon a poor ferment, bread may be sour when it comes from the oven, or it may readily undergo an unwholesome fermentation upon ageing.

Bread that is twice baked, "pulled bread" and zwiebach are at times to be preferred, for, requiring more mastication and ensalivation in order to be easily swallowed, these preparations often agree better than ordinary bread.

Also, these twice baked breads, if properly made, may be given moistened or softened in hot milk or water without becoming tenacious or sticky. When a patient has the necessary strength and willingness to chew properly it is undoubtedly better for him to take bread dry; but enfeebled patients, lacking the energy and will to masticate thoroughly, may take with advantage that which has been thoroughly saturated and perfectly softened. Even granting that bread when buttered is less easily ensalivated than when taken unbuttered, the stimulating effect to the gustatory sense which is produced by fine butter or other savory accessories more than compensates for this. Of course there are cases in which it is undesirable to excite the secretions by pleasing the sense of taste; in such cases dry bread may be the more advisable.

Experience teaches that most traditional practices in diet are based upon truth; therefore some hesitation is felt in criticising unfavorably the widespread and venerable habit of feeding dyspeptics on toast. The objections to dry toast are that it often has a hard and insoluble surface and a sticky or gummy interior. carbonized surface may stimulate the stomach unduly, while the adhesiveness of the inside of toast may be beyond the hydrating power of the ptyalin to cope with in the fundus. In the form of buttered toast, bread becomes vet more indigestible to a weakened stomach. Dipped toast and milk toast may be hard to digest unless the bread from which it is made and the method of the preparation are such that it is uniformly pulpified and easily separated in the mouth, and it must not be encumbered by an excessive richness of butter or cream. An excellent preparation for some cases is that known in some regions under the sobriquet "Yankee toast." It is made from perfectly dry bread, broken into small morsels, placed in a small bowl and covered with boiling water till thoroughly soft. The water is then drained off and a little salt and butter spread over the surface of the bread which is served hot.

There is a marked disparity in quality of the preparation known as "zwiebach." Some specimens are admirable, not too rich, crumbling almost to a powder in the mouth and devoid of particles that are hard to masticate; others possess opposite and undesirable qualities.

The use of fresh bread for the dyspeptic has been berated but the same criticism does not necessarily apply to hot stale bread; or rolls, dipped in hot water and slightly re-baked, may prove to be more digestible and more appetizing than cold bread.

Cereals are largely prescribed for the dyspeptic and

sometimes injudiciously, often indiscriminately. To be easily digested the cereals must be very thoroughly cooked and they must not be so adhesive as to interfere with ready ensalivation. The various dry kinds, prepared by manufacturers and sold in cartons, sometimes indefinitely kept in stock, are not to be recommended for the patient having a delicate digestion, nor is the conventional oat meal mush the best form of carbohydrate food for the dyspeptic. In fact, cereals are best entirely omitted from the dietary of those who have very sensitive stomachs.

When the digestive powers have improved, cereals may be used for the sake of variety. Rice, rightly prepared, is one of the best forms of carbohydrate food and should be largely employed in the diet of stomach diseases. It is, however, unsuitable if poorly prepared, not sufficiently boiled, too long boiled and soaked, or so treated that it is served in cake-like masses.

The gruels are held in deservedly high estimation by the physician experienced in prescribing for weak stomachs. A gruel should be thoroughly boiled and when served should be smooth, free from lumps, hot and delicately flavored with salt and perhaps butter.

Wheat, rice, barley, corn and oaten flour, all may be used, but not without discrimination, in making gruels. Wheat flour deserves to stand at the head for making milk gruel. This preparation is often served as an unappetizing and rather disgusting mess. Considerable art is required for its successful preparation, an art that old-time cooks seem to possess more commonly than do their modern successors.

I have a predilection for the so-called flour-ball gruel, already referred to in these pages. It is made by tying tightly in a muslin bag a pound or two of wheat flour.

This is boiled unceasingly for five or six hours. The bag is then stripped off, leaving a hard ball which should dry for a few hours. A part of the shell-like surface is broken off and the half dextrinized interior is grated and used for making the gruel.

Cornmeal, barley, rice, arrowroot, and oatmeal are preferable for making the water gruels.

Toast water is at times tolerated by a resentful stomach when gruels and other food are rejected.

For convalescents the purées made from potato, wheat flour, rice, etc., sometimes reinforced by a raw egg, stock or beef juice are valuable because of their relatively high caloric content and because of their palatability.

The convalescent sometimes enjoys rocahout prepared as follows: Take equal parts of powdered sugar, powdered cocoa, fine oatmeal and rice flour. Mix well together. Stir a tablespoonful of this into a little cold milk, then add enough hot milk to fill a cup. More or less of the mixture may be added according to the taste of the individual.

It is difficult to keep an open mind, while disturbed by the propaganda for and against its use, on the suitability of alcohol in stomach diseases. In this country most physicians have decided against the necessity, if not against the usefulness, of alcohol of all forms. This position is a conservative and in most instances a wise one; yet there are exceptional cases in which alcohol is decidedly useful.

In achylia gastrica, when it may be difficult to procure the assimilation of nutriment in sufficient amount, alcohol seems to better a patient's general health. A sound and well-matured claret or sauterne may serve a double purpose in cases of anacidity with debility.

In case of gastric atony, without hyperchlorhydria, a

teaspoonful of cognac may benefit the patient materially. When there is temporary motor insufficiency, such as often succeeds a too full meal taken when the patient is fatigued, relief generally follows the taking of a teaspoonful of brandy or a little crème de menthe or anisette.

In cancer of the stomach when a patient has distaste or even disgust for all other aliment, he will occasionally take beer, ale or stout with satisfaction. Anorexia may be so persistent that temporary stimulation of appetite is desirable. For this purpose, a tablespoonful of vino vermouth, wild-cherry brandy or kirsch may serve an excellent purpose.

On the other hand the use of liquors and all sorts of alcoholics are often causative of digestive disturbances; the habitual use of alcohol commonly occasions gastritis; taking alcohol in any form is harmful in case of peptic ulcer, gastritis, pyloric stenosis, hyperchlorhydria, hyperesthesia and in most stomach diseases. The non-alcoholic extracts of malt are useful as foods and appear to assist digestion in some way besides by their feeble diastatic properties.

Coffee and tea are often unsuspected causes of disturbed digestion. In numerous dyspeptic conditions their use should be interdicted; however, occasionally either coffee or tea may be given with benefit. When this is true, it is usually in cases where there is feeble circulation or depressed innervation, yet in other cases showing such defects these beverages cause irritability and do harm. When we are not certain of their beneficial action tea and coffee should be omitted.

Chocolate and cocoa are also of uncertain value in stomach diseases. They surpass tea and coffee in nutritive value; occasionally they agree well with the patient and help to relieve the monotonous diet that is too frequently indicated. Their use must be regulated by careful observation.

The simple infusion (tisane) made from camomile, linden flower or mint is harmless and as a substitute for tea satisfies the desire for hot drinks.

The drinking water selected for the dyspeptic is of importance. Owing to the presence of soluble minerals in considerable quantity in many waters their absorption is retarded and digestion is delayed or disturbed. Some waters, although holding mineral constituents, are absorbed quickly and appear to favor digestion.

Soft waters are generally acceptable and often even in large quantities do not encumber the digestion. Sparkling waters in moderate amount serve to stimulate gastric motility, and therefore to promote digestion in suitable cases. On the other hand when the stomach is in an irritable state, carbonated waters are objectionable. Some patients are disturbed by waters that agree well with others; although the reason for these different effects is not always evident, experience should be the guide. The action of waters upon the liver and the lower digestive tract may account somewhat for these effects upon the stomach.

The question is often asked as to when and how much water should be drunk. It must be said that this depends upon various factors, some not directly connected with digestion, for instance, upon a state of hydremia, polyuria or impeded circulation.

In healthy individuals, thirst should be a guide; however, it is not always reliable, because habit, perhaps artificially created, is more dominant. The quantity of water to be imbibed may be regulated in some by the amount of urine voided while the individual is moderately active in a temperature of about 70° F. It is better that water, for the greater part, be taken on waking in the morning or between meals; only a little being taken while eating.

This rule must be modified according to the state of the stomach. In case of hyperchlorhydria it is advisable to drink water freely at meals; in case of low acidity and decreased amylolitic or proteolitic power, water should be avoided at meals.

Excessive drinking of water may accompany the habit of excessive use of salt, disappearing when salt is taken in moderation.

Rectal Alimentation.—Rectal alimentation is by some practitioners held to have value only in proportion to the amount of water that is absorbed from the clyster, but experienced clinicians generally believe that in addition to water various foods are absorbed by the colon in amount sufficient to sustain a patient temporarily.

Preceding the introduction of a nutrient clyster, the colon should be cleared by a lavement of boric acid solution. Milk at body temperature is most commonly selected for the nutrient enema; this may be reinforced by a raw egg with or without sugar, wine or brandy.

Peptonized milk, in my experience, serves no better purpose than fresh warmed milk. A teaspoonful of thoroughly boiled flour, liquified by the diastatic action of two teaspoonfuls of extract of malt, may be added to the milk when there is believed to be a special need of carbohydrates. Special formulae advised by several recognized clinicians are given below.

Ewald recommends the ensuing mixtures as suitable for nutrient clysters:

(1)	Cognac,	(old) 30	gm.	
	Glucose	20	"	(3v)
	Water	,,	"	(žvise)

(2)	Red wine       80 gm.       (3iiss)         Glucose       20 " (3v)         Water       200 " (3viss)
(3)	Yolk of 2 eggs       20 " (3v)         Farina       20 " (3iv)         Red wine       15 " (3iv)         Chlorid of sodium       3 " (gr. xlv)
	Laudanum
Fleine	r recommends a mixture consisting of:
	Beef tea
Von L	eube has advised:
Fresh pan Warm Wa	craped beef chopped fine
Boas 1	has advised:
Into	k

## Jaccoud has recommended:

be irritable.

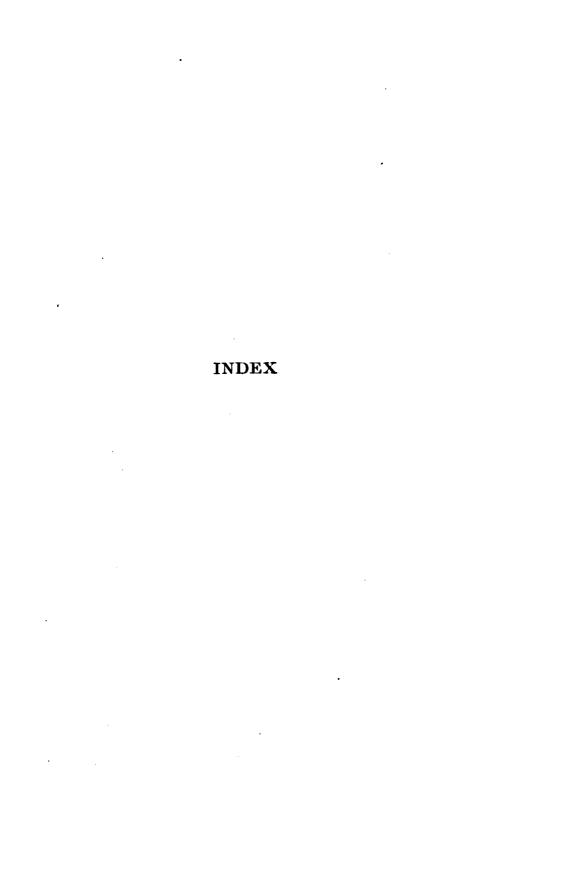
Red wine .....

Bouillon250	gm.	(3viii)
Wine120	~u	(3iv)
Yolk of 2 eggs	"	(3ii)
Peptone 5 to 20	"	(3i-3v)

to which may be added four or five drops of laudanum if the rectum

1 tablespoonful

All nutrient enemata should be warmed to body heat and introduced slowly by the means of a simple funnel connected with a short tube which terminates in a hard rubber tip.



	•		

A Achylia gastrica, cont. nature of, 604, 608 Abdomen, peculiar sensitiveness phase of, 609 of, 649 relationship between erosion of sensitiveness of, in different ingastric mucosa and, 609 dividuals, 649 slight gastric defect with, 610 Abdominal aorta, palpation of, 41 stomach contents in, 523 sympathetic ganglia of, 41 structural change in, 607 Abdominal massage, 530 treatment of, 612 deep kneading in, 552, 553 Acid gastritis, 573 effleurage in, 551, 553 Acid salts, total acidity, Töpfer's percussion over stomach in, 552 method for quantitative espetrissage in, 551 timation of, 51, 52, 53 Acidity, degrees of, 157, 158 position of patient in, 551 pressure in, 552 total indications of, 70 shaking in, 552, 553 Acidosis, 640, 646 surface friction in, 551 in diabetes, 216 vibration in, 552, 553 Acids: acetic, 54 vibratory pressure in, 552 butyric, 54 Abdominal pain, and tenderness, hydrochloric, 449 649 lactic, 53 estimation of importance of, Acne, influence on, of diet and 649 digestion, 222 Actinomycosis of stomach, 468 location of source of, 649, 652 Abscess, in limited perforation, prognosis of, 469 356, 357 symptoms of, 468 Acetic acid, determination of, 54 treatment of, 469 Acetonemia, 646 Acute catarrhal gastritis, applica-Achylia gastrica, 168 tion of term, 559 classes of, 607 clinical course of, 562 clinical course of, 607 diagnosis of, 565 etiology of, 608 etiology of, 560 functional derangements in, 608 extending to duodenum, 564 stomach contents in, 564 history of, 604 in diabetes, 215 symptoms of: constitutional disindicated by consistency turbances, 562, 563 stomach contents, 49 fever, 562, 563 indicated by means of stomach local manifestations, 564

treatment of, 566, 567

tube, 49

Acute catarrhal gastritis, cont.	Alimentation, cont.
formulas in, 566, 567	chocolate and cocoa, 725
Adenocarcinoma, 398, 399, 403	coffee and tea, 725
Adenoma, in stomach, 398, 399	eggs, 717
Adenomata of stomach, 464	fish. 716
Adhesions, from pericholecystitis,	fluid diet, 713
indicated by X-ray shad-	fruits, 719
ows, 94	gruels, 723, 724
perigastric, resulting from	meats, 715
ulcer, 292	milk diet, 714
Adrenalin, action of, on nerve	toast, 722
systems, 106	vegetables, 718
in asthma, 200	water, 726
in gastric spasm, 179, 183, 659	calories, number of assimilable
in peptic ulcer, 328, 330	in, in different patients, 710
Aerophagy, 522	care to be exercised in, 706, 707
cause of, 522	denutrition from standpoint of
eructations in, 522, 523	general economy in, 710
symptoms of, 522	depression of metabolic power
treatment of, 524	in, by over-fatigue, 769
Age, consideration of, in peptic	dyspeptics' fear of certain ar-
ulcer, 229	ticles of food in, 712
for cancer, 379, 386	ethnological importance of, 707 formulation of diet with proper
Akoria, 195	distribution of calorie val-
treatment of, 196	ues, 711
Alcohol, gastritis due to, 600	general adoption of rational
in cancer, 386, 387	diet in, 708
susceptibility to, 601 use of, 724, 725	importance of knowledge of di-
Alcoholic gastritis, 559	etetics in, 709
symptoms of, 601	laboratory experimentation in,
treatment of, 602, 603	709
Alcoholism, causing gastritis, 561	
"Algesimeter," 42	over-indulgence in, 712
Alimentary canal, pouches of:	poor, evil results of, 707
cecum, 116	proposed dietaries for, 708
stomach, 116	question of fluid or solid food
representation of position and	in, 713
movement of, by radio-	rectal, 727
graphic shadows, 83	repletion from standpoint of
Alimentary intoxication, 108, 109	metabolism, 710
relationship between gastritis	semi-fluid diet in, 713
and, 110	tables representing proportion
Alimentation, articles of diet:	of proteids, fats, and car-
alcohol, 724, 725	bohydrates, 711, 718
breads, 720, 721	varied diet m, 719
cereals, 722, 723	Alkalies, use of, in diabetes, 216
•	

Alkalinity of blood, theory of, as Appendicitis, abdominal pain in, protection against gastric 657 luice, 248 as cause of dyspepsia, 122, 123 differentiated Alkaloids, use of, in peptic ulcer, chronic, from peptic ulcer, 308 314 Anacidity, 167 gastric disturbances secondary Anadenia gastrica, 574 to, 122 Anaphylaxis, 108, 109 due to cecal stasis, 117, 118 clinical manifestations of, 110 stomach symptoms of, 122 dangers from, in use of serum, Appetite, in diagnosis, 26 330 loss of, in anorexia nervosa, 191 dangers of, 109 Arrhythmia, due to gastric disturbance, 210, 211 Anasarca, with cancer, 426 Anastomosis, for hourglass stom-Arteriosclerosis, 615 case cited of, with pain after ach, 360 Anemia, absence of gastric secreeating, 616 tion in, 604, 605, 606 causing peptic ulcer, 258, 259, with cancer, 424, 425, 426 260 Angioneurotic edema, abdominal diagnosis of, 618 pain due to, 656 treatment of, 618 Angor abdominalis, tenderness Arteritis, probable syphilitic case with, 655 cited of, with absence of ar-Anodynes, danger of, in obscurterial sclerosis symptoms, ing the real nature of con-617 dition, 352 Arthritis, due to gastro-intestinal in cancer pain, 456, 459 disease, 219 Anomalies of secretion, 156 effect of eating on, 219 Anorexia, treatment of, in canstomach and other complicacer, 456 tions in, 219 Anorexia nervosa, loss of appestomach in relation to, 218 tite in, 191 Ascites, with cancer, 426, 427 nature of, 191 Asthma, due to gastric disturbprognosis of, 192 ance, 200 symptoms and signs of, 191, effect of eating on, 200 192 Asthma dyspepticum, distinguishtreatment of, by drugs, 194 ing features of, 201, 202 form of, 201 dietary, 193 disciplinary, 192, 193 prognosis of, 202 symptoms of, 201 formulas for, 193, 194 soothing mixtures for, 193 treatment of, 202 Antacids, use of, in peptic ulcer, use of term, 200, 201 Atonic dilatation of stomach, 502 313, 314, 315, 360 Antipepsin, deficiency in, prelim-Atonic stomach, indicated by Xinary to ulcer, 257 ray shadows, 95 Atony, gastric, 183 Antitrypsin, 448 Antitryptic reaction in cancer, indicated by stomach tube, 69, 447 71

Atony, cont. indications of, 49 use of term, 102 Atropin, action of, on nerve systems, 105, 106 in asthma, 200 in gastric spasm, 179 in peptic ulcer, 329, 342, 350 in treatment of ulcer, 203, 266 Auerbach's plexus, 11 Auscultation, deglutition sounds by, 45 Autodigestion, 247, 266 treatment of, in peptic ulcer, 327 Auto-intoxication, 108 Autonomic system of nerves, action of, 104

## В

Bacilli, Oppler-Boas, 62, 63 Bacteria, intestinal, in cecum, 116 Bacterial infection, causing peptic ulcer, 253, 254 Bassler's rubber bulb, in diagnosing cardiospasmus, 672 Bassler's sieving pail, for lavage water, 376 Beatson's theory of cancer, 378 Beaumont, William, on trauma in peptic ulcer, 249 Belladonna, in peptic ulcer, 314, 324, 337 Benign tumors of stomach: 463 adenomata and polypi, 464 false tumors, 465 fibrous polyps, 465 inflammatory masses, 465 polyadenomata, 464 polypi, 464 Benzidine test for occult blood, 61 Bernard, Claude, experiments of, on resistance of tissue to action of gastric juice, 247, 248

viduals, 561 Bile, indications from, by stomach tube, 72 Bilious attacks, due to liver, 111 Bilocular stomach, 290, 291, 358, 359 diagnosis of, 359 treatment of, 360 Bismuth, and chloroform, in peptic ulcer, 316 in colon, 84 in duodenum. 93 in peptic ulcer, 313, 315, 317, 318, 324, 349 in stomach, 84 radiographic shadows produced by, 83, 84 weighing down of stomach by. in radiography, 84 Biuret test, for peptones, 55 Bladder, distended. associated with dyspepsia, 129 stone in, associated with dyspepsia, 129 Block, on peptic ulcer, 253 Blood, 60 in cancer, 451 in stomach, acting as emetic, indications from, by stomach tube, 71 occult, Benzidine test for, 61 Guiaiacum test for, 60 Blood supply; arterial, 10 capillary, 10 veins. 11 Blumer, on tuberculosis of stomach, 208 Boas, "algesimeter" of, 42 method of, for quantitative estimation of rennin and its zymogen, 59 on acute gastrectasis, 517 test of, for motor activity of stomach, 65 Bread, 720, 721

Berries, effect of, on certain indi-

Bronchitis, effect of eating on. Cancer, cont. 199, 200 distribution of, urban and Buccal cavity, digestion within, 1 rural, 382, 383 Bulimia, 189, 195 due to gastric ulcer, 362 treatment of, 196 early diagnosis of, importance Burns, superficial, duodenal or and difficulty of, 379, 380 gastric ulcer from, 223, 264 edema with, 426 theories regarding, 295 external agent causing, 378, 379 Butyric acid, determination of, 54 fever as a symptom in, 424 forms of, as shown by coun-C tries, 385, 386, 387 inaccessible and difficult to Cachexia, diagnostic importance diagnose, 389 of, in cancer, 427 Calcium chlorid, in peptic ulcer, frequency of, 379, 381 330, 331 gelatinous carcinoma, 403 Calcium lactate, in peptic ulcer, geographical distribution of, in Bavaria, Kolb, with table 330, 331 showing, 392, 393 Calomel, in peptic ulcer, 325 Caloric food values, 150, 711 geologic influence on, 379 grafted upon pyloric ulcer, con-Camphophenique, in peptic ulcer, tingent with pyloric ste-315 Cancer, absence of laboratory nosis, 412 growing upon ulcer, 414, 416, proof in, 422, 423 age for, 379, 380 alcohol, influence of, on, 386, growth of, from ulcer scar, 415 387 on chronic peptic ulcer, 416, anasarca with, 426 on recurring peptic ulcer, anemia with, 424, 425 418, 421 anorexia in, 456 importance of early diagnosis bleeding in, 475, 476 of, 454 colloid, 403 increase of, in country, 382 comparison of cases of, per million living in Sud- and inflammatory tumor resembling, Nordbayern, 390, 391 case cited, 413 complications deceiving diagliving cases of, per million, in nosis of, 380 Switzerland, table of, 394 cure of, 453 localization of, as to organs afdiagnosis of, 379, 380 fected, 383, 384, 385 location of, among cases in Engdifficulties of, in some cases, 422, 423, 424 land, 389 surgical exploration in, 454 medullary, 403 diagnostic importance of occult metastases, consideration of, in, blood test in, 477 404 differential diagnosis of, 412, mortality from, 382, 393, 395 413, 414 in England, 388 distinguished from chronic cain Germany, per million liv-

ing on Jan. 1st of year, 394

tarrhal gastritis, 586

Cancer, cont.	Cancer, cont.
of digestive tract, 386	glycyltryptophan test for,
percentage of cases of, 397	447, 448, 449
of gastro-intestinal tract pre-	hemolytic action of blood
dominating, 386	serum, 447
of peritoneum, 386	lessening of secretions in, 441
origin of, 378	Oppler-Boas bacilli, 441
pain in, treatment of, 456	in stools, 450
pathology of, 397, 398	squamous-celled, 403
preceding ulceration, 415	statistics of localization as to
predilection of, for organs ad-	organs affected, 383
jacent to orifices for re-	statistics of prevalence of, 381
ception of food and air,	surgery for, importance of
386	early diagnosis, in, 410, 411
predispositions to, 397	success of, in pyloric region,
predominance of, in stomach	409
over other tumors, 397	symptoms of, 380, 381
progressive increase of, 379	theories of cause of, 378
in Munich, 394	toxemia in treatment of AEC
radiography for, 414	toxemia in, treatment of, 456
recovery from, 452	treated as neurasthenia, 423
recurrence of, 453	treatment of, anorexia in, 456 by diet, 457
relative increase of, 395	by hygienic care, 458
relative susceptibility to, at va-	medical, 453
rious age periods, 389, 392	pain in, 456
scirrhous, 403	surgical, 453, 454, 455
special diagnoses of, 441	toxemia in, 456
albuminuria in, 449	vomiting in, 455
antitryptic reaction in blood	ulceration of, 415
serum, 447, 449	vomiting in, treatment of, 455
by appearance of blood in	Cancer en nappe, 407
stomach contents, 441	Cancer of esophagus, 690
bacterial flora in stomach con-	diagnosis of, by character of
tents in, 450	deglutition murmur, 692
blood in, 451	by esophagoscope, 691
by blood count, 449	by radiography, 691
by eancer nests in stomach	by stomach tube, 691
contents, 441	preponderance of, in males, 690
by cutaneous reaction, 447	treatment of, by gastrostomy,
by finding of tumor cells, 442,	692
445	by radium (Einhorn), 692
by microscopic study of stom-	by stomach tube, 692
ach contents, 441, 442, 445	by surgical removal (Willy
by Roentgen rays, 452	Meyer and Franz Torek),
by Salomon's method, 445	692, 693
disappearance of hydro-	Cancer, of intestines and rectum,
chloric acid secretion, 441	386
,	<del>-</del>

Cancer of liver, accompanying Cancer of stomach, cont. symptoms of, 427, 428 failure of gastric secretion jaundice with, 427, 428 in, 432 Cancer, of organs of reproducgastric atrophy in the aged, tion, 388 in, 432 Cancer of pylorus, 409 general facts in, 436 frequency of, 410 in old people, absence of gaspicture of average case of, 411 tric secretion in, 432 primary, contingent with pyincreased rigidity of stomach loric stenosis, 412 in, 434 Cancer of stomach, 388 location of tumor in, 433 abdominal pain in, 429, 658 masked cases of, 440 adenocarcinoma, 403 over-tonicity in, 434 age for, 400 palpation in, 433, 434 anatomy of, 400 physical examination in, 433 appearance of, 400 , ptosis in, 436 ascites with, 426, 427 diagnostic importance of local cachexia with, 427 edema in, 435 cells in, 402 of pancreatitis in, 436 of phlegmasia alba dolens, character of, 404 chronic gastric catarrh of, 402 435 classification of, 402 of umbilical phlebitis in, 435 as to character, 404 diffuse, 404, 407 as to location, 404 dropsy with, 426 clinical history of, 404 general considerations of, 378 diagnosis of, 431 hematemesis with, 427 advanced symptoms in, 432 hemorrhage with, 401, 427, 432 complicated by anemia, 440 histology of, 402 indicated by X-ray shadows, complicated by ascites, 436 100 complicated by infection from pathogenic organisms, 437 intestinal symptoms obscuring complicated by invasion into diagnosis of, 428, 429 thorax, 437 introduction to, 378 complicated by metastases, invasion of thorax by, 437 435, 437, 438, 439 involvement of other organs by, complicated by resemblance 400, 401 to obscure ulcer, case of, location of, 404 437, 438, 439 at anterior or posterior wall complicated by resemblance of stomach or lesser curvaof syphilis to, 440 ture, 408 confusing conditions in, 433 at cardiac end of stomach, consideration of liver in, 435 consideration of pancreas in, in pyloric region, 409 435, 436 invading interior of stomach, detection of onset in, 431 407 melena with, 427 enlargement of glands a facmetastases with, 426, 427, 429, tor in, 436 435, 437

Cancer of stomach, cont. pain in, 433 pathology of, 378, 397, 398, 399 percentage of cases of, 397 perforation with, 401 physical examination in, 433 predominating in women, 395, 396 prognosis of, 452 pyloric stenosis due to, 401 statistics showing, 396 symptomology of, 404 symptoms of, general, 408, 409, local, 407, 408, 409, 410 ulceration with, 401 unusual forms of, 422 varieties of, 402, 403 vomiting in, 432 Cancer of stomach, liver and pancreas, death by, per million, Carbohydrates, mastication of, 1 Carcinoma, cylindrical-celled, 403 gelatinous, 403 medullary, 403 Oppler-Boas bacilli of, 62, 63 Solomon's test for, 61 scirrhous, 403 squamous-celled, 403 tryptophan test for, 61 Carcinoma ventriculi, 396 Cardia, position of, relative to fundus, 4 relaxation of, in digestion, 2, 3 spasm of, 179, 180 Cardiac depression, due to gastric disturbances, 210 Cardiac disease, stomach in relation to, 210 treatment of, for gastric symptoms, 211, 212 Cardiospas-Cardiospasm, see mus. Cardiospasmus, 179, 180 analysis of cases of, 673, 674 eases cited, 673, 674

gradually appearing symptoms, 677 Mikulicz operation not successful, 677 minor attack of, with preceding history of cerebrospinal fever and neurasthenia, and subsequent severe attack, two sudden attacks of, 674, 675 delay in deglutition murmurs of, 671 diagnosis of, by esophagoscope, 672by physical examination, 671 by a sound, 672 by stomach tube, 671, 672 by symptoms, 671 differential diagnosis of, 673 location of, 670 morbid anatomy of, 670 pathological physiology of, 670 prognosis of, 181, 182, 678 symptoms of, 671 symptoms and signs of, 181 treatment of, 182 by dilatation, 679, 680 by esophagoplication, 681 by gastrostomy, 680 by psychotherapy, 681 Carlsbad water, for constination

Cardiospasmus, cont.

in peptic ulcer, 318, 325, 333

Carminatives, formula of, for motor disturbances, 174, 175

Cases cited, of cardiospasmus, 673, 674

Case No. XXVIII, arteriosclerosis, with pain after eating, 616

Case No. XXII, cancer following on recurring peptic ulcer and long-continued chronic dyspepsia, 418, 421

- Cases cited, cont.
  - Case No. XXV, cancer of stomach, mistaken for duodenal ulcer and cholelithiasis, 437, 438, 439
  - Case No. XXIV, cancer of stomach, with profound secondary anemia, 424, 425, 426
  - Case No. XXIII, cancer, with absence of laboratory proof, slight local evidence, striking derangement of general health, 422, 423
  - Case No. XXXVI, cardiospasmus, gradual development of, with slowly increasing symptoms for years, and subsequent severe attack, 675, 676
  - Case No. XXXV, cardiospasmus; minor attack of, with preceding history of cerebrospinal fever and neurasthenia, and subsequent severe attack, 675
  - Case No. XXXVII, cardiospasmus, symptoms appearing gradually; sticking of food; pain during meals; pain referred to dorsal spine, cure, 677
  - Case No. XXXIV, cardiospasmus, two sudden attacks of, 674, 675
  - Case No. XXXIII, cyclic vomiting in childhood, 645
  - Case No. XVI, duodenal ulcer, perforating, special points of interest in, 300, 301
  - Case No II, dyspepsia, cure of, by lithotrity, 129
  - Case No. III, dyspepsia, cured by catheterization, 129
  - Case No. I, dyspepsia cured by operation, 126

Cases cited, cont.

- Case No. XVII, floating kidney with symptoms of peptic ulcer, 309
- Case No. VII, on trauma of associated with pulmonary tuberculosis, 203
- Case No. XXX, gastric syphilis, 622
- Case No. XXXI, gastric syphilis, mistaken for cancer, 623
- Case No. XXXII, gastric syphilis, mistaken for cancer of liver, 624
- Case No. XII, gastric ulcer, recurring acute, near pylorus, 271
- Case No. XXVII, gastritis, resulting from pyloric stenosis and gastrectasia, 565
- Case No. XXVI, gastrosuccorrhea, 485
- Case No. VIII, hysteria and gastric ulcer, 262
- Case No. XXI, inflammatory tumor resembling cancer, 413
- Case No. IV, morbid fear of drinking water, associated with gastric symptoms, 130
- Case No. VII, on trauma of stomach, 249, 250
  - Case No. XV, peptic ulcer, chronic, ending in cancer, 288, 289
  - Case No. XI, peptic ulcer, recent, near lesser curvature, 270
  - Case No. XIV, peptic ulcer, recurring, 287
  - Case No. XVIII, peptic ulcer symptoms, none found on exploration, 310
  - Case No. XXIX, probable syphilitic arteritis, absence of arterial sclerosis symptoms, 617

740

"Cecum mobile," and dyspepsia, Cases cited, cont. Case No. V, pyloric spasm, 178, 179 Case No. XXXVIII, rupture of the esophagus by vomiting. Previous health of the patient, good, 694, 695 Case No. XIX, surgery for chronic peptic ulcer, 345 Case No. XX, treatment before operation in pyloric stenosis due to gastric ulcer, 366 Case No. X, ulcer and dermatitis, multiformis, 263 Case No. IX, ulcer and neuralgia, 263 Case No. XIII, ulcer at cardiac end of stomach, 272 Castor oil, in peptic ulcer, 325 Catarrhal gastritis, 557, 558 acute. See Acute catarrhal gaschronic. See Chronic catarrhal gastritis. indicated by stomach tube, 71 Catheterization, dyspepsia cured by, Case III cited, 129 Cecal stasis, and dyspepsia, 116 appendicitis due to, 117, 118 chronic, 118 colon bacillus infection in, 120 course of a mild attack of, 118 diagnosis of, 118 due to appendicitis, 118 gastric disturbances due to, 118 mistaken for migraine, 119 new seizures of, 117 recurrence of, 120 symptoms of, 117, 118, 119 treatment of, general, 119 medical, 120 surgical, 120 Cecum, anchoring of, 118 intestinal bacteria in, 116 motor insufficiency of, 116 overmotility and displacement

of, 116, 117

116, 117 Cells, chief, 8, 9 columnar epithelial, 7, 8 parietal, 8, 9 secreting, 8 Cereals, 722, 723 Cerebral vomiting, 633 Cerium oxalate in peptic ulcer, 313, 317, 327 Charcoal, for motor disturbances, 174 Childhood, effect of hardships during, 25 Chloroform, in peptic ulcer, 316 with bismuth, in peptic ulcer. 316 Chlorosis, with gastric ulcer, 248 Chocolate and cocoa, 725 Cholecystitis, and dyspepsia, 113 chronic. differentiated from peptic ulcer, 307 gastric symptoms of, 113, 114 treatment of, 115 vomiting of, 635 Cholelithiasis, chronic, differentiated from peptic ulcer, 307 Chronic catarrhal gastritis, 572 clinical course of, 579, 580 diagnosis of, 585, 586, 587, 588 diagnosis of, by exclusion, 580 by microscopic examination. by physical examination, 583 diagnostic errors in, 585 differential diagnosis of, 585, 586, 587, 588 etiology of, 577 gastric juice in, 582 morbid anatomy of, 572 mucosa in, 582 prognosis of, 588, 589 relapses in, 584 symptoms of, 579, 580 tongue in, 583 treatment of, by alkalies, 598 by diet, 591, 592, 593, 594

Chronic catarrhal gastritis, cont. by drugs, 596, 599 by lavage, 595, 599 by mineral acids, 596, 597 by predisposing and underlying causes of, 590 direct, 591 Chyme, acid, passing into duodenum, 18 mixing of, in stomach, 17 Circulation, effect of eating upon, Circulatory depression causing dyspepsia, 127, 128 Cirrhosis, 403 Cirrhosis ventriculi, 576 Cirrhotic gastritis, 576 "Coagulose" in peptic ulcer, 330 Coffee, 725 Coffee grounds vomit, 476 Cohnheim, "cell rests" theory of, on cancer, 378 description of palpation by, 36, 37 on hyperchlorhydria as cause of peptic ulcer, 256 on trauma of stomach, 249 Colds, effect of eating on, 199 "Colica mucosa," abdominal pain in, 658 Colloid cancer, 403 Colon, abdominal pain in affections of, 658 location of, determined by percussion, 44 radiography in study of, 84 Colon bacilluria, symptoms of; Colon bacillus infection, in cecal stasis, 120 Colon massage, 553 Connor, Lewis A., on acute gastrectasis, 515, 516 Constipation, in diagnosis, 28 in peptic ulcer, 317, 318, 324 Contents of stomach; See Gastric contents

"Corde-colique," 38 Corpus luteum, extract of, for dyspepsia attending menopause, 134 Coryza, effect of eating on, 199 Cruveilhier, erosions of, 239 on cause of peptic ulcer, 250, 251 Cruveilhier's disease, 238 Curvatures, lower, position of, 5 upper or lesser, position of, 5 "Cutaneous reaction" of cancer, 447 Cyclic vomiting, 643, 646 of childhood, 644, 645 treatment of, 647 Cytotoxic influences on peptic ulcer, 255

# D

Debout d'Estrées, on oxaluria, 136 therapeutics of, principle of, 138 Deglutition, impeding of, 2 in esophagus, 667, 668 mechanism of, 2 Deglutition murmurs, delay of, in cardiospasmus, 671 Deglutition sounds, 45 by auscultation, 45 de la Tourette, Giles, on peptic ulcers in hysterical, 262 Depressive neurosis, 105 Dermatitis, in children, 221 Dermatitis multiformis, associated with peptic ulcer, 263 in relation to nervous system, 263 Diabetes, acidosis in, 216

digestion in early stages of, 214 disturbed metabolism in, 214 drinking of water in, 215 gastric chemistry of, reports on, 216

gastric functions in, 214, 215

Diabetes, cont.	Diagnosis, cont.
gastric intolerance in, 216	percussion in, 43
stomach in, 216	personal history in, 24
Diagnosis, 24	physical examination in, 30
auscultation in, 45	preceding illnesses in, 25
	radiography in, 83, 94
appetite considered in, 26	regurgitation in, 28
change in residence or occupa-	
tion considered in, 25	sensitiveness to pressure in, un-
childhood influences considered	der palpation and percus-
in, 25	sion, 41
constipation in, 28	sleep in, 29, 30
difficulties and importance of,	special avocations considered in,
in cancer, 410, 411	25, 26
difficulties of, in some cases of	stomach tube, dangers of, in,
cancer, 422, 423, 424	73
of dyspepsia and eye-strain,	diagnostic value of, in, 72,
142, 143, 144	73
exploration of abdomen for purposes of, 123	use of, in, 67, 68, 69, 70, 71,
fluoroscopy in, 83	stools in, 29
habits of patient in, 26	string test in, 77
headache in, 30	syphilis in differential, 621
insomnia in, 29	vomiting in, 27, 28
inspection of patient in, 31	Diaphragmatic hernia, causing
mistakes in, of dyspepsia and	gastric obstruction, 362
eye-strain, 141	Diathesis, dyspepsia associated
of malaria and gastric symp-	with, 135
toms, 224	gastric symptoms of, 135
of motor disturbances and	lithemia in, 135
gas, 170, 171, 172	oxalemia in, 137
nausea in, 27	oxaluria in, 136
nervous system considered in,	Diet, in cancer, 457
29	in gastric ulcer, 335, 336
of dyspepsia and eye-strain,	during convalescence, 338,
difficulties of, 142, 143,	339
144	for ordinary cases, 336, 337,
mistakes in, 141	338
of dyspepsia, and local disease	Lenhartz on, 334
of stomach, 153	milk in, 334, 336
of epigastric pain or distress,	von Leube on, 333
27	in hyperchlorhydria, 159, 163,
	164
of eructation, 28 of gastric ulcer complicated	
with gastrosuccorrhea, 348,	in malaria, 227
349	in psoriasis, 223
	influence of, on acne, 222
of hyperchlorhydria, 161, 162 of peptic ulcer, 305, 306	varied, importance of, 719
	Dietetics, importance of knowl-
palpation of patient in, 33	edge of, 709

743

"Dietl's crisis," 214 Dieulafoy, erosions of, 238 Digestion, amplebatic, 17, 22	Dilatation of stomach, cont. acute, 502 anatomical changes due to, 504
Digestion, amylolytic, 17, 22	
deglutition of food in, 2	atonic, 501, 502
effect of, on cells of stomach, 9	clinical course of, 504, 505
effect on, of structural diseases	confusion of, with motor insuf-
of heart, 211	ficiency, 500, 501
functional derangements of,	diagnosis of, 507
154	etiology of, 502, 503
gastrie, 72, 123	from stenosis, 501
hydration of food in, 1	gastric tetany in, 512
in pylorus, 17	gastromegaly in, 501
in stomach, 15, 16, 17	objective symptoms of, emacia-
influence of, on acne, 222	tion, 507
kitchen, influence of, on, 1	secondary to obstruction, symp-
mastication, importance of, in,	toms of, 505
1	symptoms of, 504, 505
mechanism of, 22	use of term, 500
motor function of, 2	vomiting in, 506
process of, 1, 2	Dilated and distended right heart,
psychic influences on, 1	pain and tenderness in,
skin diseases in relation to,	656, 657
220	Dimethylamidoazobenzole test for
	free hydrochloric acid,
Digestive abnormality, importance	
of, in disease, 151	50
Digestive disturbances, due to	Diseases, stomach in relation to,
faulty teeth, 1	199
due to nervous system, 104	Distended bladder, associated with
due to respiratory tract dis-	dyspepsia, cure of, by
eases, 2	catheterization, 129
due to vagus innervation de-	Distress, epigastric, in diagnosis,
rangement, 2	• 27
"Digestive gastrosuccorrhea," 481,	Diverticula of esophagus, diagno-
485	sis of, 697
diagnosis of, 486	treatment of, 697
particularities of, 486	Diverticulum formation, 669
symptoms of, 486	Dolbey, on lymphangitis as cause
treatment of, 487	of peptic ulcer, 251, 252
Digestive symptoms, applicability	"Drip sheet rub" in inducing
of, to gastric syphilis, 625	
	sleep, 194
Digitalis, action of, on nerve sys-	Dropsy, with cancer, 426
tems, 105	Drugs, action of, on nerve sys-
elimination of, 212, 213	tems, 105, 106
Dilatation, indicated by stomach	selective action of, in individu-
tube, 71	als, 106
Dilatation of stomach. See Gas-	use of, in control of hemor-
trectasis.	rhage, 479, 480

Duodenal alimentation. See "Duodenal feeding."	Dyspepsia, etc., cont. arising from disturbance of
indicated for peptic ulcer, 321	sense of smell, 139
Duodenal cap, 93, 94	associated with diathesis, 135
"Duodenal feeding," 79, 80	associated with distended blad-
description of apparatus for,	der, 129
80	associated with disturbances of
	hearing, 140
process of, 80, 81	
technique of, 81	of internal secretion, 133, 134
uses of, 81	
Duodenal ulcer, as complication	of sight, 140
in infections, 296	of special senses, 139
classification of, with gastric	associated with eye-strain, 140,
ulcer, 298	141, 142, 143
clinical history of, 303	associated with gastritis, 145
coincident with cholecystitis,	associated with gastrorrhagia,
303	pyloric spasm, jaundice,
contingent with pyloric steno-	operative cure of, 126
sis, 412	associated with hyperchlorhy-
due to superficial burns, 223,	dria, gastric distress, urica-
264	cidemia, stone in bladder,
theories regarding, 295	cure of by lithotrity, 129
duodenal feeding in, 81	associated with lithemia, 135
in newborn, 294	associated with menopause, 134
incidence of, 301, 302	associated with neurasthenia,
indicated by radiographic shad-	129, 130, 131
ows, 98	associated with oxalemic gout,
influence of sex in, 302	137
melena neonatorum in, 294	associated with oxaluria, 136,
infants with, 294	137
motor insufficiency in, 304	associated with pregnancy, 134
nature of, 294	associated with pregnancy, 134 associated with tuberculosis.
pain in, 304	131, 132, 133, 203
perforating, 304	cecal stasis and, 116
case cited of, 300, 301	"cecum mobile" and, 116, 117
lobar pneumonia and acute	chronic, latent gout with, 135
pleurisy mistaken for, 305	confused with alcoholic gastri-
recognized by string test, 303	tis, 601
simulating congenital stenosis	cure of, by catheterization, case
of pylorus, 294	III cited of, 129
uremia in, 296	by lithotrity, case cited of,
vomiting in infants with, 294	129
Duodenum, bismuth in, for radio-	by operation, case cited of,
grams, 93	126
position of, 6	diagnosis of, considerations in,
Dyspepsia, and cholecystitis, 113	144
appendectomy for relief of, 122	diet, articles of, as cause of, 111

Dyspepsia, etc., cont. discussion of term, 101 due to appendicitis, 122, 123 due to circulatory depression, 127, 128 due to eye-strain, 123 due to functional disorders of gastric digestion, 123 due to heart disease, 127 due to liver disturbance, 111 due to local disease of stomach, due to occupations, 127 due to other than gastric disorders, 122, 123, 126, 128 due to urinary tract irritation. 128 explanation of symptoms of, functional, associated with gastritis, 145 in hard-working men, 127 irritative symptoms of, differentiated in peptic ulcer and hyperchlorhydria, 274 misconception of term, 154 nature of, 145, 146 nervous disharmony as cause of, 103 nervous origin of symptoms of, 107 operative cure for, case cited, 126 origin of symptoms of, 106 oxalemic, symptoms of, 137 palliative measures in, dangers of, 152 "pretubercular," 204 sympatheticotonic states in, 155 symptoms and signs of different types of, 155 theories of causes of, 102, 103 treatment of, 146 accompanied by cholecystitis, by "tracking" disease, 151 caloric food values in, 150

Dyspepsia, etc., cont.

danger of palliative measures
in, 152
dieting in, 149, 150
exercise in, 148
local disease of stomach in,
153
proper living in, 148, 149
rest in, 148, 149
source of trouble in, 147
special conditions in, 153
super-alimentation in, 149
vitality of patient in, 147, 148
vagotonic states in, 155
Dysentery, duodenal feeding in,
81

### E

Eating, appetite in, 26 causing gastritis, 560 comfort following, 27 effect of, on colds, 199 on furunculosis, 222 on gout, 219 increase of pain by, 660 indiscreet, causing fever, 227 effect of, on heart, 210, 211, 212 effect of, on skin, 221 respiratory disorders due to, laryngeal irritation after, 199 prescribed, in cardiac disease, 211, 212 psychic state in relation to, during pulmonary tuberculosis, 206, 207 relief of pain by, 660 Ecchymosis, hematemesis with, 472 Edema, with cancer, 426 Edkins, on gastric juice, 20 Effleurage, 551 Eggs, 717 Einhorn, dilatation of pylorus by,

Einhorn, etc., cont.	Erythremia, hematemesis with, 473
"duodenal feeding" of, 79, 80	Esophageal neurosis, 670
erosions of, 239	Esophagitis, 698
gastric erosions of, 372	acute, 698
"string test" of, 77	causes of, 699
Elsner's modification of Mett's	chronic, bleeding in, 700
method for quantitative es-	causes of, 700
timation of pepsin, 57	nature of, 700
Emetin, use of in hemorrhage, 480	symptoms of, 701
Endomyces albicans, 467	treatment of, 701, 702
Enemata, use of, in peptic ulcer,	clinical history of, 699
326, 333	diffuse purulent, 699
Enteroliths, 466	due to infection, 698
Enteroptosis, conformations of	symptoms of, 699
victims to, 536	treatment of, 700
due to faulty physical develop-	Esophagoscope, in diagnosing car-
ment, 532	diospasmus, 672
due to heredity and acquired	Esophagus, action of, in diges-
causes, 536	tion, 2
of Glénard, 530	anatomy of, 666
postural defects in, 523	cancer of, 690
prevalence of, 537	cicatricial stricture of, diagno-
spinal deformity in, 533, 534,	sis of, 684
535	treatment of, by dilatation, 685
Enzymes, active secretion of, 70	Sippy's method, 686, 687,
deficiency in secretion of, 70	688, 689
Epigastric hernia, as cause of	corrosive destruction of mucosa
peptic ulcer, 261	of, 702
Aaron on, 261	deformity of, results of, 669
pain due to, 656	deglutition in, 667, 668
Epigastric pain or distress in di-	diverticula of, diagnosis of, 697
agnosis, 27	treatment of, 697
Epithelium, 63	fibers of, 667
Ergot, use of, in peptic ulcer, 328	foreign bodies in, 703
Erosions, follicular, 242	function of, 666
gastric, 372	functional disease of, 702
homorphagia 949 942	hemorrhage of, 704
hemorrhagic, 242, 243	treatment of 705
hemorrhagic infarct, 244	treatment of, 705
in furrows, 244	idiopathic dilatation of. See
lenticular type of, 244	Cardiospasmus.
of Dieulafoy, 238	invasion of, from morbid pro-
of peptic ulcer, 238	cesses of neighboring struc-
according to Hayem, 239, 240	tures, 666, 667
	minor spasm of, 669
Cruveilhier's, 239	
Einhorn's, 239	mucosa of, 667
Eructation, in aerophagy, 522, 523	muscular coat of, 667
in diagnosis, 28	normal tonicity of, 2

Esophagus, action of, cont. Exercises, prescribed for gastropobstruction of, 668 tosis, 542 by peptic ulcer, 682, 683 "Exulceratio simplex of stomach," by typhoid ulcer, 682 238 depending conditions of, 668 Eye-strain, dyspepsia associated due to conditions within, 669 with, 140, 141, 142, 143 due to congenital defects, 681, dyspepsia due to, 123 effect of, 487 due to infectious gastrosuccorrhea due to, 487 granulomata, 688 vomiting due to, 636 due to pressure, 668 due to structural diseases, 681 F due to traction, 668 False tumors, 465 treatment for, 683 unusual forms of, 690 Fasting, in peptic ulcer, 323, 335, paralysis of, 702 peptic ulcer at lower end of, Fasting stomach, fluid in, 47 "Fat necrosis," of Baker, 245 peristalsis of, 667 Favus of stomach, 468 physiology of, 666 Febrile conditions, See Fever. rupture of, spontaneous, 693 Fecal vomiting, 636 Fermentation, 523 traumatic, 693 sensory disturbances of, treatin diabetes, 215 indicated by stomach tube, 72 ment for, 702 spasm of, 669 disturbances mistaken stricture of, cicatricial, 683 for, 171, 172 structure of, 666 Ferments, 54 syphilitic infiltration of, 682 Fever, effect on digestion of, 217 tonus of, 667 in acute catarrhal gastritis, 562, 563 typhoid ulcer of, 682 Estimations, quantitative, of hystomach condition in, 217 drochloric acid. Sahli treatment of stomach in, 218 Fish, 716 method, 51 of hydrochloric acid, Töpeffect of eating, on some indifer's method, 51 viduals, 560 Floating kidney, of pepsin, 55 differentiated of rennin and its zymogen, from peptic ulcer, 309 Flour-ball gruel, 723 Eucalyptol, use of, in peptic ul-Fluid diet, 713 cer, 315 Fluoroscopy, 83 "Enzone," for hyperchlorhydria, advantages of, 87, 88 cinematographic reproductions 165 Ewald test meal, 48 of bismuth shadows by, 88 Examination, microscopical, 62 dangers of, 87 personal history, 24, 25, 26, 27, diagnostic value of, 87, 88 28, 29, 30 movements of stomach reprophysical, 30 duced by, 88

Follicular erosions, 242 Fomentations, use of, See Poultices. Food, articles of: bread, 720, 721 cereals, 722, 723 chocolate and cocoa, 725 coffee and tea, 725 eggs, 716 fish, 716 fruits, 719 gruels, 723, 724 meats, 715 milk, 714 purées, 724 rocahout, 724 toast, 722 vegetables, 718 zweiback, 722 causing gastritis, 560 course of boluses of, in diges-	Formulas, cont. for rectal alimentation, 727, 728 for soothing mixtures for anorexia nervosa, 193 for vomiting, 638 for vomiting, in peptic ulcer, 327 gastric sedatives Nos. 1 and 2, 164, 165 Friction, stethoscopy associated with, 46 Fruits, 719 Functional disorders, of digestion, 154 of stomach, symptoms and signs of different types of, 155 Functional disturbances, anomalies of secretion, 156 Fundus, 4, 539 nerve control of, 21 position of, 4
tion, 2, 15, 22 desire for, in bulimia, 195 in polyphagia, 195 effects on individuals of certain, 109, 110, 111	Furunculosis, effect of eating on, 222  G
for rectal alimentation, 727, 728 idiosyncrasies to certain, 109, 110, 111 unwholesome, urticaria induced by, 222 Food poisoning, 108 "Food vomiting" in gastric ulcer, 285 Foreign bodies, in esophagus, 703 in stomach, 466 "Formes frustes," of Graves' dis-	Gall-bladder, palpation of, 39 Gas, death from overdistension of stomach with, 180 in stomach, 18, 19, 522 indications of excess of, by stomach tube, 72 motor disturbances mistaken for, 170, 171, 172 radiographic shadows produced by distension of a part by, 83 Gaseous distension, 522
ease, 134  Formulas, carminative, for motor disturbances, 174, 175  for alkalies in gastritis, 598  for chronic esophagitis, 702  for gastritis, 566, 567  for hyperesthesia in peptic ulcer, 313, 314, 315, 317  for mineral acids, for gastritis, 597	Gastralgia, causes of, 196 differentiated from peptic ulcer, 307 in cholecystitis, 115 in malaria, 223 nature of, 196 prognosis of, 197 symptoms and signs of, 197 treatment of, 198

Gastrectasis, See Dilatation of stomach. acute, 515 appearance of, after laparotomy, 519 classes of, 517 death from pressure on heart in, 518 description of condition in, 517, 518 distension of stomach in, 518 due to aerophagy, 522 origin of: abdominal shock, 519 distension, 516, 517, 518 obstruction, 515, 516 various, 517 radiography in diagnosis of, 519 symptoms of, 517, 518, 519 treatment of, 519, 520 diagnosis of, by palpation, 509 by stomach tube, 510, 511 differential diagnosis of, 512 due to gastric ulcer, 364 due to stenosing gastritis, 361 duodenal feeding in, 81 gastric tetany in, 526 hypertrophy of muscle coat in, 507 in relation to arthritis, 218 indicated by X-ray shadows, 96 objective symptoms in, tension and torsion, 509 prognosis of, 512 treatment of, 513 Gastric acidity, estimation of, from stomach contents and not from vomitus, 278 Gastric atony, 183 clinical course of, 493 diagnosis of, 494, 495 due to leak of electic tiesue, 490 due to leak of electic tiesue, 490 due to leak of electic tiesue, 490	Gastric atony, cont. fluoroscopy in betraying, 495 hormones in, 490 in relation to arthritis, 218 in relation to dilatation, 492 in relation to gastric secretion, 491 in relation to motor insufficiency, 488 insomnia in, 494 overeating and drinking as cause of, 491 prognosis of, 496 psychic influences in, 491 radiograms showing, 492 symptomology of, 493 temporary, 493 treatment of, by diet, 497 by hygienic measures, 496 by medicaments, 498 Gastric contents, 47 acids of, 49 blood in, 60 chemistry of, 49 color of, 48 consistency of, 49 ferments in, 54 fluid in, in fasting stomach, 47 free hydrochloric acid in, 49 juice in, 47 mucus in, 60 odor of, 48 test meals and, 48 zymogens in, 54 Gastric crises, 625, 626 abdominal pain of, 659 in early tabes, 626 treatment of, 627 Gastric derangement. aggravation of renal disease by, 214 Gastric digestion, functional disorders of, 123 retardation of, indicated by stomach tube, 72 Gastric dilatation serving of 62
of parathyroid glands, 526 due to lack of elastic tissue, 489	stomach tube, 72 Gastric dilatation, sarcinæ of, 63
due to weak or degenerated mus- cles, 490	Gastric distress, associated with dyspepsia, 129

Gastric hyperesthesia, cont. Gastric distress, etc., cont. symptoms of, 185, 186 cure of, by lithotrity, case cited, treatment of, drugs in, 188 129 general and local measures in cholecystitis, 115 relieved by swallowing of food, in, 187, 188 Gastric irritation, vomiting due to, theories of causes of, 102, 103 Gastric juice, causes of stimula-Gastric disturbances, asthma due tion of, 19, 20 to, 200 classification of, 101, 102 chemical composition of, 19, 47 due to cecal stasis, 118 description of, 19 excited by hormone, 608 due to eye-strain, 123 due to neuropathic causes, 125 loss of digestive power by, 611, manifestations of, 612 individual 102 persistent secretion of, 481 "jumping at conclusions" in diresistance of tissues to action of. 247, 248, 266, 267 agnosis of, 123 mistaken use of surgery for, secretion of, 156 123, 124, 125, 126 secured from pigs, 613 secondary to chronic appendispecific enzymes of, 8 citis, 122 stimulation of, by foods, 20 Gastric elimination, of toxic maby psychic influences, 19 terial, 213 Gastric motion, diminished, 183 Gastric erosions, classification of, Gastric mucous membrane, substance of, 7 as disease, 373 Gastric mycosis, 467 nature of, 372 Gastric neurosis, 104, 105, 154 of Einhorn, 372 organisms found in pus of, 373, abdominal tenderness in, 665 due to functional disorders in pathological changes of, shown wide sense of term, 130 by photomicrographs, 375, treatment of, 130 376 Gastric obstruction, due to diapost-mortem studies of, 372, 373 phragmatic hernia, 362 pyorrhea alveolaris with, 373 treatment of, in peptic ulcer, symptomology of, 377 322 tissue fragments recovered from Gastric secretion, absence of, in wash of, 374, 375 pernicious anemia, 604, 605, treatment of, 377 606 mechanism of, 20, 21 differentiated Gastric erythism, from peptic ulcer, 309 mixing of, with food, 16, 17 Gastric hemorrhage, stools in, 477 modifications in, 606 symptoms of, prior to hematepersistent absence of, 604 mesis, 478 Gastric sedatives, 318, 333 "Gastric hormone," 20, 21 prescriptions Nos. 1 and 2, for Gastric hyperesthesia, 70, 185 hyperchlorhydria, 164, 165 etiology of, 185 Gastric spasm, abdominal pain with, 659 prognosis of, 186

Gastric spasm, cont.	Gastric syphilis, cont.
adrenalin for, 179, 183	gastric crises in, 625
Gastric stagnation, reflex bron-	infrequency of, 621
chial irritation from, 200	involvement of stomach by, di-
Gastric stasis, following pyloric	rect or indirect, 620
spasm, 177	method of elimination as possi-
Gastric stenosis, causing dilata-	ble diagnosis in, 622
tion, 501	modes of attack of, 620
Gastric stimuline, 613	modes of expression of, 620
Gastric symptoms, associated with	palpable tumor from, 621
disturbance of special	paroxysmal gastralgia with, 621
senses, 139	possible application of digestive
associated with morbid fear of	symptoms to, 625
drinking water, 130	Gastric tetany, 512, 526
associated with neurasthenia,	clinical picture of, 527
129, 130, 131	diagnosis of, 528
associated with pulmonary tu-	origin of, theories on, 526
berculosis, 203, 204	prognosis of, 529
due to colon bacilluria, 121	treatment of, 529
due to fever, 217	by calcium, 526
due to vascular disease, 616	Gastric tuberculosis, classification
in appendicitis, 122	of, 208
in cardiac disease, 210	findings on examination in, 208,
in cecal stasis, 119	209
in cholecystitis, 114	transmitting of infection in, 208
in diabetes, 214, 215	ulcerative form of, 208
in functional heart trouble, 128	Gastric ulcer, chronic, distin-
in Graves' disease, 133	guished from chronic ca-
in malaria, 223	tarrhal gastritis, 588
in surgical exploration for can-	classification of, with duodenal
cer, 454	ulcer, 298
in tuberculosis, 131, 132	complications of: gastrosuccor-
of diathesis, 135	rhea, 347
of pregnancy, 134	perforation, 350
outstripping of usual symptoms	pyloric stenosis and obstruc-
by, in Graves' disease, 134	tion, 360
Gastric syphilis, 620	consideration of, in relation to
case cited of, 622	gastrie syphilis, 620, 621
mistaken for cancer, 623	diet in, 335
mistaken for cancer of liver,	in convalescence, 338, 339
624	in ordinary cases, 336
consideration of, in relation to	Lenhartz, 334
gastric ulcer, 620, 621	von Leube, 333
diagnostic importance of pain	due to arteriosclerosis, 258, 259,
in, 625	260 .
distinguished from chronic ca-	due to herpes, 268
tarrhal gastritis, 588	due to superficial burns, 223
- ,	•,,

•	
Gastric ulcer, etc., cont.	Gastric ulcer, etc., cont.
duodenal feeding in, 81	vomitus in, 285
gastritis in, 281	with abrupt onset, 286
hematemesis in, 282	Gastric unrest, 170
hematin in, 284	or spasm, application of term
hemorrhage from, 474	of, 102
hemorrhage in, fatalities from,	treatment of, in peptic ulcer,
283	322
percentage of, 282	Gastric varix, 575
significance of, 283	Gastritis, acid, 573
treatment of, 479	acute catarrhal, See Acute ca-
hyperchlorhydria in, evolution	tarrhal gastritis.
of, 279, 280	acute phlegmonous, See Acute
indicated by X-ray shadows, 96	phlegmonous gastritis.
influence of sex in, 303	alcohol in, 578
lactic acid in, 280, 281	alcoholic, 600
melena in, 282, 283	anadenia gastrica, 574
mortality of, by von Leube, 333	as complication of general in-
occult blood in, 284	fections, 561
pain after ingestion of certain	associated with functional dys-
foods in, 275	pepsia, 145
pain in, character of, 275, 276	case cited, resulting from pyloric
direction of, 276	stenosis and gastrectasia,
due to idiosyncrasy, 275	565
effect of alkalies on, 277, 278	catarrhal, indicated by stomach
effect of pressure on, 276, 277	tube, 71
effect of psychic states on,	chronic catarrhal, see Chronic
277	catarrhal gastritis.
localized, 275	cirrhotic, 576
radiation of, 276	classification of: acute, 559
rectal alimentation in, 331, 332,	alcoholic, 559
340	catarrhal, 557, 558
recurring, 286, 287	chronic, 559
recurring acute, near pylorus	etiological, 559
case cited, 271	interstitial, 557, 558, 559
sequelae of: 347	irritative, 559
cancer, 362	mucous, 557, 558
fistula, 358	parenchymatous, 557, 558, 559
gastrectasis, 361	
hourglass stomach, 358	phlegmonous, 559 sclerotic, 559
perigastritis, 358	secondary or sympathetic, 559
treatments for: Lenhartz, 334	toxic, 559
Von Leube, 332	complication of uremia in, 561
unknown element in etiology of,	depending on syphilis, 412
267. vomiting in, 284, 285, 286	diagnosis of, care and time re-
significance of, 286	quired for, 557
aignineance or, 200	damen ror, oo.

Gastritis, acid, cont.	Gastroptosis, cont.
difficulties in, 555, 556	diagnosis of, by auscultation,
not dependent on symptomol-	<b>541</b>
ogy alone, 556	by palpation, 541
positive, by means of stom-	by radiography, 541
ach contents, 556	due to child bearing, 531
presumptive, 556	due to tight wearing apparel,
due to alcoholism, 561	530, 531
due to cardiac disease, 212	due to weakness of abdominal
due to diabetes, 215	ligamentous supports, 531
due to infections, 561	exact position of stomach in, 541
due to intoxications, 212, 213	intra-abdominal pressure in, 532
due to uremia, 578	position of stomach in, 43
general considerations of, 555	predisposition to, in certain in-
glandular, chronic, symptoms	dividuals, 531
of, 70	prevalence of, 538
hematemesis from, 475	in adults, 530
in gastric ulcer, 281	in women, 530, 531
interstitial, 573	relief from, 542
involvement of muscle coats in,	relief in, 540
490	symptoms of, 540, 541
polypoid, 577	treatment of, by medical gym-
predisposition to, in certain in-	nastics: 542
dividuals, 560	breathing exercises (as-
relationship between alimentary	sistive), 547
intoxication with metabolic	correct standing, 543
disturbance and, 110	creeping position, 546
stenosing, 574	deep breathing, 543
symptoms of cancer and ulcer	exercise for abdominal
in, 556	ptosis and too prominent
symptoms of dyspepsia in, 555	lumbar anterior curve,
toxie, 568	<b>54</b> 3
treatment of, in malaria, 227	exercise for obliterated lum-
in tuberculous patients, 207	bar curve and pronounced
with peptic ulcer, 246	dorsal posterior curve,
Gastro-diaphane, use of, 75	546
Gastro-enterostomy, relief and	lateral trunk exercises, 548
healing of pyloric and duo-	leg elevation, 545
denal ulcer by, 257	lying on back, both knees
use of, in gastrosuccorrhea, 349	lifted to chest, 545
Gastrojejunostomy, for peptic ul-	lying on back, one knee
cer, chronic, 346, 347	lifted to chest, 544
Gastroliths, 466	postures to be held, 549
Gastromegaly, 501	sideways bending, 548
Gastroplasty, for hourglass stom-	trunk rotation (twisting),
ach, 360	549
Gastroptosis, causes of, 530	physiologic, 542
- · ·	• • • •

Gastrorrhagia, 470, 471 associated with dyspepsia, operative cure of, 126 causes of, 471, 472 clinical use of term, 474 in absence of ascertainable lesions, 472 operative cure of, case cited of, 126 symptoms of, 475 Gastrosuccorrhea, 167, 279, 481 care in diagnosis of, 348 characteristics of, 348 digestive, see Digestive gastrosuccorrhea. eye-strain causing, 487 in relation to gastric ulcer, 347 treatment of, 349 Gastrosuccorrhea continua, Riegel, etiology of, 481, 482 prognosis of, 483 symptoms of, 482, 483 Gastrosuccorrhea periodica (Rossbach), 481, 484 abdominal pain of, 659 case cited of, 485 psychic influences in, 484 symptoms of, 484 Gastroxynsis, 484 Gelatinous carcinoma, 403 General peritonitis, due to perforating ulcer, 353 Geographical distribution, consideration of, in peptic ulcer, 229 Germain Seé Test meal, 48 "Girdle pain," of tabes, 626 Glands: peptic, 7 pyloric, 9 Glandulae agminatae, 10 Glandulae lenticulares, 10 Glandular enlargement, in cancer, 436 Glandular gastritis, symptoms of, by stomach tube, 71

Glénard, "corde-colique," described by, 38 on enteroptosis, 530 Glycyltryptophan test, of cancer, 447, 448, 449 Gout, latent with chronic dyspepsia, 135 oxalemic, associated with dyspepsia, 137 Graves' disease, gastric symptoms of, 133, 134 gastro-intestinal trouble in, 133 symptoms of, 133, 134 treatment of, 134 Gross's method, for quantitative estimation of pepsin, 57, 58 Gruels, 723, 724 Guiaiacum test, for occult blood, Günzburg's solution, to determine free hydrochloric acid, 49, Gymnastics, medical, for gastroptosis, 542

### H

Habitus enteroptoticus, 40 Hairballs, 466 Hamamelis, use of, in peptic ulcer, 328 Hammerschlag's method, for quantitative estimation of pepsin, 55, 56 Hayem, erosions according to. 239, 240 Headache, in diagnosis, 30 Hearing, digestion affected by disturbances of, 140 Heart, causing dyspepsia, 127 effect on digestion of structural diseases of, 211 "pounding of," due to gastric causes, 210 relationship between stomach and, 210

Hematemesis, 470	Hepp, Maurice, securing of gas-
as symptom of gastric ulcer,	tric juice by, 613
329	Hernia, diaphragmatic, 362
causes of, 472, 473	retroperitoneal or Treitz, differ-
diagnostic importance of, in	entiated from perforating
cancer, 427	ulcer, 355
from bleeding within stomach,	under palpation, 41
471	Herpes, gastric ulcer due to, 268
from gastritis, 475	History of patient, points in, 24,
in gastric ulcer, 282	25, 26, 27, 28, 29, 30
in lieu of catamenial discharge,	Hormones, gastric juice excited
473	by, 608
in syphilis, 472	_stimulating peristalsis, 490
with bleeding outside stomach,	Hourglass stomach, 290, 291
470, 471	diagnosis of, 359
with ecchymosis of gastric mu-	following gastric ulcer, 358
cosa, 472	indicated by stomach tube,
with erythremia, 473	69
with polycythemia, 473	treatment of, 360
venesection for, 474	"Hunger pain," 304, 412, 660
Hematin, in gastric ulcer, 284	of duodenal ulcer, 348
Hemolytic action, in cancer, 447	Hydrastin hydrochlorate, use of,
Hemorrhage, control of, by drugs,	in peptic ulcer, 328
479, 480	Hydration, of amalaceous mate-
diagnostic value of, in cancer,	rial, 1
427	process of, 1
due to vasomotor disease, 473	Hydrochloric acid, quantitative es-
in cancer of stomach, 432	timation of: Sahli method, 51
in gastric ulcer, 474 fatalities from, 283	Töpfer's method, 51
percentage of cases of, 282	secretion of, 8
significance of, 283	total acidity, Töpfer's method
in peptic ulcer, 328	for quantitative estimation
indicated by stomach tube,	of, 51, 52, 53
71	Hydrochloric acid (free), dimeth-
of esophagus, 704	ylamidoazobenzole test for,
sera in control of, 479	50
symptomology of, 477	Günzburg's solution test for, 49,
thirst accompanying, 331	50
treatment for, in gastric ulcer,	qualitative test for, 49
479	resorcin solution test for, 50
in peptic ulcer, 327, 328, 329,	tests for determination of,
330	qualitative, 49
significance of, 328	Hydrocyanic acid, in peptic ulcer,
Hemorrhagic erosions, 242, 243	327
Hemorrhagic infarct, of peptic	Hygiene, in cancer, 458
ulcer, 244	Hyperacidity, 157

Hyperacidity, cont. indication for treatment of peptic ulcer, 312 Hyperchlorhydria, 157 abdominal pain with, 659 action of internal secretion in. 160 application of term, 102 associated with arthritis, 119, associated with dyspepsia, 129 associated with peptic ulcer, 255, 256, 312 cure of, by lithotrity, case cited, 129 diagnosis of, 161, 162 differential diagnosis of, 161, 162 due to gastric ulcer, 364 due to kidney disease, 214 etiology of, 158 functional, contingent with pyloric stenosis, 412 indicated by stomach tube, 70 irritative dyspepsia of, distinguished from that of peptic ulcer, 274 prognosis of, 162 reality of functional origin of, 421 relative hypotonia with, 160 symptoms and signs of, 159, 160 treatment of: diet, 163, 164 general, 166 gastric sedatives, Nos. 1 and 2, 164, 165 irrigation of stomach, 166 nascent hydrogen peroxid, 165 prescriptions, gastric sedatives Nos. 1 and 2, 164, 165 salines, administration of, 164 therapeutic indications, 162 vagotonic states in, 159 Hyperemesis gravidarum, theory of, 641

Hyperemia, congestion of liver as cause of, 111 of liver, 113 Hyperesthesia, application of term, 102 gastric, 185 with peptic ulcer, 312 Hypermotility, 170, 183 symptoms and signs of, 171 Hyperorexia, 195 treatment of, 196 Hypersecretion, 167 estimation of, by calculation of total stomach contents at a given time, 65 indicating treatment for peptic ulcer, 312 indications of, by consistency of stomach contents, 49 by Ewald test meal, 48 in fasting stomach, 47 Hypochlorhydria, 167 diagnosis of, 168, 169 differential diagnosis of, 168, 169 etiology of, 167 prognosis of, 169 treatment of, 169, 170 use of term of, 102 Hypomotility, treatment of, 184 Hypotonia, relative, with hyperchlorhydria, 160 Hysteria, associated with gastric ulcer, 262

# Ι

Ice, use of, in peptic ulcer, 327, 329, 334, 368
Iced drinks, gastritis due to, 561
Ichthyol, use of, in peptic ulcer, 313
Idiopathic hematoma auris, and peptic ulcer, 263, 264
Idiosyncrasy, 560
instances of, 109
nature of, 108

Ileus, vomiting of, 635 Indigestion, discussion of term, 101 Infections. causing gastritis, 561 duodenal ulcer as complication in, 296 Inflammation, as cause of peptic ulcer, 250, 251 Inflammatory tumors, deep-seated, 413 resembling cancer, 413 Innervation of stomach, extrinsic, 11 intrinsic, 11 Innutrition, in diabetes, 215 resulting from gastric ulcer, 358 Insomnia, due to gastric atony, 494 in diagnosing, 29 Inspection of patient, points to be noted in, 31, 32, 33 position assumed in, 31 Internal secretions, peptic ulcer due to derangement of, 257, 258 Interstitial gastritis, 557, 558, 559, 573 Intoxications, alimentary, 108, 109 of digitalis, 212, 213 Intragastric pressure, 12, 13, 14, 17 Iodoform, use of, in peptic ulcer, 313, 324 Ipecac, use of, in peptic ulcer, 327 Irregular secretion, indication for treatment of peptic ulcer. 312 Irritable stomach, indicated by Xray shadows, 95 Irritative gastritis, 559 Irritative neurosis, 104 Ischochymia, See Motor Insufficiency. in diabetes, 215

J

Jaundice, with cancer of liver, 427, 428 with dyspepsia, operative cure of, 126

K Kelling's test for determining lactic acid, 54 Kemp, on acute gastrectasis, 515, 516 Kidney, left, position in relation to fundus, 4 palpation of, 39, 40 Kidney disease, see Renal. Kinnicutt, on gastric tetany, 526, 527 Kneading, deep, in massage, 552, 553 Kussmaul, on gastric tetany, 526 L Lactic acid, tests for determination of: Kelling's, 54 Uffelman's, 53 Lang, point of, 662 Laryngeal irritation, due to eating, 199 Larynx, disease of, affecting digestion, 2 Latent gout, with dyspepsia, 135 Lavage, in cancer, 455, 456 in diabetes, with gastric symptoms, 215 in peptic ulcer, 323, 324, 328, 330, 349, 368 in pulmonary tuberculosis, 207 Lead colic, abdominal pain in, 655, 656 Lenhartz treatment, for gastric ulcer, 334 Lenticular erosions, 244 Lignol, use of, in peptic ulcer,

313

Limited perforation of stomach, 355, 356, 357, 358, 359 diet in, 227 Liquid paraffin, in peptic ulcer, 327 Liquid vaselin, use of, in peptic ulcer, 327 Lithemia, with dyspepsia, 135 latent, 224 Lithotrity, dyspepsia cured by, case II cited, 129 225 Liver, abdominal pain in passive congestion of, 657 cancer of, 427, 428 hyperemia of, 113 metabolism and, 111, 112 palpation of, 38, 39 torpid, 111, 112 Loeper, on disturbances of internal secretion as cause of peptic ulcer, 258 on internal secretion, 160 on oxalemia, 137 Loeper and Esmonet, on abdominal pain and tenderness, Loss of substance, treatment for, in peptic ulcer, 327 Lugol's solution, for starch digestion test, 59 Lumbar backache, rest in bed for, Lymphangitis, as cause of peptic ulcer, 251, 252 in peptic ulcer, 236 Lymphatics, 10 Melena, 477 McBurney's point, 662 McGuire, on simultaneous spasms of cardia and pylorus, 678

McGuire, on simultaneous spasms of cardia and pylorus, 678
MacCallum, on gastric tetany, 526
Magnesium carbonate, use of, in peptic ulcer, 317
Magnesium sulphate, use of, in peptic ulcer, 325
Malaria, aestivo-autumnal type, prolongation of gastric irritability in, 224
treatment of, 226

Malaria, cont. diagnosis of, 224, 225 digestive derangement in, 224 digestive manifestations of, 224 liver and spleen in relation to, stomach in, 223 treatment of, 224, 226 gastritis in, 227 vomiting in, 224 Mal de mer, 638 Malignancy, syphilis mistaken for, 623, 624, 625 Malignancy of stomach, indicated by stomach tube, 69 Malnutrition, in diabetes, 215 Mann, on gastroptosis, 530 Massage, abdominal, 530, 551 of colon, 553 Mastication, faulty teeth and, 1 value of, in digestion, 1 Mastoiditis, gastric symptoms accompanying, 140 Mathieu and Remond, "Remnant test" of, 65 Matthes, on trauma of stomach, Matzinger, on othematoma, 264 Mayo, on surgery for cancer, 453 Median line, 4 Medical gymnastics, 542 Medullary carcinoma, 403 Meissner's plexus, 11 diagnostic importance of, in cancer, 427 in gastric ulcer, 282, 283 Meltzer, on deglutition sounds, 45 Membrane propria, 9 Metabolic peculiarities, 135 Metabolism, and liver, 111 disturbed in diabetes, 214, 215 peculiarities of, 135

subject of, 109

Metastases, 404

Metastases, cont. Motor disturbances, cont. diet in, 174 with cancer, 426, 427, 429 with sarcoma of stomach, 461, Mett's method, modified by Elsner, for quantitative estiin, 173 mation of pepsin, 57 Microscopical examination. for Oppler-Boas bacilli, 62, 63 Motor inhibition, 15 Migraine, cecal stasis mistaken for, 119 Milk diet, value of, 714 500, 501 Morbid fear of drinking water associated with gastric sympin cecum, 116 toms, 130 Moritz' test for motor activity of stomach, 63 Morphin, in general peritonitis, use of, in peptic ulcer, 329, 342 Morvan's disease, case of, 263 Motility, lack of indication of, by Ewald test meal, 48 658 Motion, disorders of, 102 importance of disturbances of, 70, 71 indications of disturbances of, of stomach, 60 Motor activity, estimation of, by calculation of total stom-Myasthenia, 492 ach contents at a given time, 65 origin of, 488 of stomach, excessive, indicated Mycelium, 468 by stomach tube, 69 strong, indicated by stomach tube, 71 12 tests for: Boas, 65 factors of, 14 Moritz, 63 function of, 12 Motor disturbances, 170 mistaken for gas, 170, 171, 172 prognosis of, 173 N symptoms and signs of, 171, 172 treatment of, anti-spasmodics in, 173 Nerve supply, 11 carminatives for, 174, 175

charcoal in, 174

hydrotherapy in, 173 sedatives in, 173 voluntary control of patient Motor excitability in peptic ulcer, treatment of, 322, 323, 324 Motor insufficiency, confusion of, with dilatation of stomach, due to vertical stomach, 358 gastric atony in relation to, 488 in pyloric obstruction, 363, 364 in pyloric spasm, 177 in pyloric stenosis, 412 in stomach, 69, 116 use of term, 102, 492 Mouth, digestion in, 2 Mucous colitis, abdominal pain in, Mucous gastritis, 557, 558 Mucous, excessive, indications of, Muscles of stomach, 12 Muscularis mucosae, 7, 8 Myasthenia gastrica, 502 "Myenteric plexus," 11 "Myenteric reflex," action of, 11,

Nauheim baths, use of, 128 Nausea, in diagnosis, 27 Nerves of stomach, over-stimulation of, 15

Nervous disharmony, as cause of Occult blood, cont. dyspepsia, 103 in gastric ulcer, 284 disturbances, distin-Nervous guished from chronic catarrhal gastritis, 586 indicated by stomach tube, excited by lavage, 70 "Nervous exhaustion" associated with dyspepsia, 130 Nervous influences, in peptic ulcer, 262 Nervous system, diagnostic importance of, in digestive disordigestive disturbances due to, 104 in relation to ulcer, 262 Neurasthenia, associated with dyspepsia, 129, 130, 131 Neuroses, affecting sensation, 185 depressive, 105 gastric, 104, 105 irritative, 104 Neurotrophic influences, in gastric ulcer, 267 in peptic ulcer, 262 Nitrates, for treatment of arteriosclerosis, 618 Normal stomachs, in radiography, 90 types of, 90 Nux vomica, use of, 128 Obrastzow's method of palpation,

Obstruction, benign, at pylorus, indicated by stomach tube, 69 gastric, 362 indicated by stomach tube, 67, of pylorus, indicated by X-ray shadow, 94 Occult blood, Guiaiacum test for, 60

Occupation, consideration of, in peptic ulcer, 229 Odors, sweat reflex arising from. 139 Old people, absence of gastric secretion in, 432 Operation, dyspepsia cured by, 126 in cecal stasis, 120 in cholecystitis, 115 Ophüls, on relation between arterial lesions and ulcer, 260 Opium, use of, in peptic ulcer, 314 Oppler-Boas bacilli, 62, 63 Orchitic internal secretion, relationship of, with digestion, 134 Orexin tannate, use of, in peptic ulcer, 327 Organic acids, total acidity of, Töpfer's method for quantitative estimation of, 51, 52, 53 Othematoma, and peptic ulcer, 264 Over-acidity, in gastric ulcer, 278 Over-secretion, due to kidney disease, 214 in gastric ulcer, 278 Over-tonic stomach, indicated by X-ray shadows, 95 Over-tonicity of stomach, 170 symptoms and signs of, 171 Oxalemia, Loeper on, 137 treatment of, 138 Oxalemic dyspepsia, symptoms of, 137 Oxalemic gout, dyspepsia associated with, 137 Oxaluria, Debout-d'Estrées 136 dyspepsia associated with, 136,

hereditary nature of, 137

Oxaluria, etc., cont.
persistent, 137
prevalence of, in America, 136
treatment of, 136, 138

P

Pain, abdominal, 649 depending on incomplete and unsuspected inguinal hernia, 652 diagnostic value of, 653 due to angioneurotic edema, due to diseased pancreas, 656 due to epigastric hernia, 656 heightened by increase of intra-abdominal pressure, 652 heightened by lateral lying posture, 652 hunger pain, 660 in angiospasm, 655 in appendicitis, 657 in cancer of stomach, 658 in colica mucosa, 658 in colonic affections, 658 in dilated and distended right heart, 656, 657 in gastric crises, 659 in gastric spasm, 659 in gastrosuccorrhea periodica, 659 in hyperchlorhydria, 659 in lead colic, 655, 656 in passive congestion of liver, in peptic ulcer, 659, 660 in peritonitis, 656 in thoracic disease, 657 in vascular crises, 655 location of seat of, 661, 662 reproduction of, for locating purposes, 661 seat of, in various affections, decrease of, by eating, 660 diagnosis of, as reflex or transmitted, 651

Pain, etc., cont. epigastric, in diagnosis, 27 local character of, 652 reflex causes of, 650, 651 transmitted, 650, 651 heightened by pressure, 653 heightened by tension, 653 in cancer, anodynes for, 456, 459 of stomach, 433 treatment of, 456 in gall-bladder, 653, 654 in peptic ulcer, source of, 316 in perforating ulcer, 351, 352 increase of, by eating, 660 of movable kidney, 654 of pelvic organs, 654 of renal calculus, 654 of ulcer, 653 previous to appearance of herpes zoster eruption, 655 Palpation, abdominal aorta under, "corde-colique" under, 38 deceit under, by scybalous mass, gall-bladder under, 39 in gastrectasis, 509 kidney under, 39, 40 liver under, 38, 39 other organs under, 41 methods of; deep finger, 35 Obrastzow's, 36, 37 superficial, 34 of hernia, 41 of lower border of stomach, 36 of spleen, 40 preparation of patient for, 33, 34 pylorus under, 37, 38 sensitiveness to pressure under, Pancreas, pain in diseases of, 656 Pancreatitis, acute hemorrhagic, differentiated from perforating ulcer, 354 chronic, symptoms of, 436

Parenchymatous gastritis, 557,	Peptic ulcer, cont.
558, 559	characteristics of, 234, 235
Paresthesia gastrica, nature of,	elinical history of, 287
189	conditions leading to, 233
prognosis of, 190	distinguished from recent, 236
symptoms and signs of, 189, 190	endarteritis in, 236
treatment of, 190	ending in cancer, 288, 289
Paroxysmal gastralgia, with gas-	glandular lesions in, 237
tric syphilis, 621	hemorrhage in, 235, 236, 237
Passive congestion of liver, ab-	microscopic changes in, 237,
dominal pain in, 657	238
Patient, auscultation of, 45	obstruction due to, 235, 236
examination of, personal his-	resemblance of, to cancer, 235
tory in, 24, 25, 26, 27, 28,	treatment of, 343, 344
29, 30	by gastrojejunostomy, 346,
physical, 30	347
inspection of, 31	surgical, 345, 346, 347
palpation of, 33	clinical course of, 270
percussion of, 43	consideration of age of patient
Pavy, theory of, on alkalinity of	with, 229
blood, 248	consideration of geographical
Pepsin, 54	distribution, 229
qualitative test for, 55	consideration of occupation in
quantitative estimation of,	patient with, 229
Gross's method for, 57, 58	consideration of sex in patient
Hammerschlag's method for,	with, 228
55, 56	constipation with, 317, 318, 325
Mett's method, modified by	cytotoxic influences on, 255
Elsner, 57	diagnosis of, 305, 306
Pepsinogen, 9	as to location, 272
qualitative test for, 55	as to location, significance of
Peptic ulcer, 228	spasm at cardia in, 289
abdominal pain in, 659, 660	diet in, see Gastric ulcer.
associated with dermatitis mul-	differentiated from appendici-
tiformis, 263	tis, chronic, 308
associated with hyperchlorhy-	differentiated from chronic
dria, 312	cholecystitis, 307
autodigestion in, 247	differentiated from chronic
bacterial infection as cause of,	cholelithiasis, 307
253, 254	differentiated from floating kid-
cardinal symptoms of, 305, 306	ney, 309
causes of, 247, 268	differentiated from gastralgia,
chronic, adhesions due to, 236	307
anatomical changes in, 237	differentiated from gastric ery-
areas of lymphangitis in, 236	thism, 309
cancer growing upon, 416,	differentiated from other forms
<del>4</del> 17	of ulceration, 228

Peptic ulcer, cont.	Peptic ulcer, cont.
differentiated from renal colic,	and contiguous parts, 341
309	irregular secretion, 312
discoloration in, 232	loss of substance, 327
	motor excitability 200 200
due to arteriosclerosis, 258, 259, 260	motor excitability, 322, 323, 324, 326
due to derangement of internal	vomiting and stasis, 322, 327
secretions, 257, 258	inflammation as cause of, 250,
due to epigastric hernia, 261	251
due to obstruction to circula-	irritative dyspepsia of, distin-
tion, 258, 259, 260	guished from that of func-
due to trophic changes, 264	tional hyperchlorhydria, 274
due to vascular obstruction, 260	
	lack of infolding of mucosa in,
duodenal, 294	251
duodenal feeding in, 81	location of, 229, 297, 298, 299,
effect of stasis on, 256, 257	300
erosions of, 238	lowered alkalinity of blood in,
Cruveilhier, 239	248
Dieulafoy, 238	lymphangitis as cause of, 251,
Einhorn, 239	252
follicular, 242	microscopic changes in, 232
Hayem, 239, 240	morbid activities making up
hemorrhagic, 242, 243	pathology of, 311
hemorrhagic infarct, 244	morbid anatomy of, 229
in furrows, 244	mortality of, von Leube, 333
lenticular type of, 244	nature of, 268
processes of, 245	nervous influences on, 262
evolution of, 246	neurotrophic influences on, 262
exterior appearance of part of	occurring in hysterical, 262
stomach involved in, 232	pain with, 316, 322
gastritis with, 246	source of, 316
	pathogenesis of, 246
hyperchlorhydria in relation to,	pathology of, 246
255, 256	
hyperesthesia with, treatment	primary lesions of, 245
for, 313	recent, bottom of, 232
in lower end of esophagus, 682,	description of, 230
683	distinguished from chronic,
in newborn, 294, 295	236
indications for treatment of:	hyperesthesia with, 312
auto-digestion, 327	induration of borders of, 230
gastric obstruction, 322	near lesser curvature, 270,
gastrie spasm, 322, 324, 325	272
gastric unrest, 322	onset of, 273
hemorrhage, 327, 328	punched-out appearance of,
hyperacidity, 312	230, 247
hypersecretion in, 312	recovery from, 232, 233
involvement of serous coat	sides of erosion of, 230
	,

	<b></b>
Peptic ulcer, cont.	Peptic ulcer, cont.
sides of opening of, 231	under pressure. 42
rectal alimentation in, 331, 332,	with abrupt onset, 286
340	Peptones, Biuret test for, 55
recurring, cancer growing upon,	Percussion, colon, location of, de-
418, 421	termined by, 44
case cited, 287	in massage, 552 method of, 43, 44
elinical history of, 286	position of patient for, 43
rest cure for, 318, 319, 320, 321 result of: hourglass stomach,	preparation for, 44
290, 291	sensitiveness to pressure under,
perigastric adhesions, 292	41
pyloric stenosis, 291	stomach position determined by,
secondary changes in, 246	43, 44
string test in diagnosis of, 77,	Perforating Ulcer, 350
78	abscess in limited perforation,
summary of pathogenesis of,	in, 356, 357
269, 270	differential diagnosis of from
symptom-complex of, 272	hernia, retroperitoneal or
symptomology of, 270	Treitz, 355
trauma as cause of, 249	from pancreatitis, acute hem-
treatment of, 311	orrhagie, 354
by correcting disturbed physi-	from torsion of ureter, 355
ology, 316	general, 353
by decrease of motor excita-	general peritonitis due to, 353
bility, 322, 326	limited perforation in, 355, 356,
by drugs, 313, 314, 315, 316	357 <b>,</b> 358, 359
by duodenal alimentation, 321	symptoms of, 351
by fasting, 323	in limited perforation, 356,
by ice-bladder, 327	357
by lavage, 323, 324	treatment of, 353
by poultices, 326	Pericholecystitis, adhesions from,
by rest, 318	indicated by X-ray shad-
details of, 320, 321	ows, 94
importance of complete,	Perigastric adhesions, resulting
318, 319	from ulcer, 292
hyperesthesia with, 313	Perigastritis, following gastric ul-
in involvement of serous coat	cer, 358
and contiguous parts, 341	Periodic vomiting, 648
indications for, 311	Peristalsis, indigestion, 22
Lenhartz, 334 pain in, 316, 322	of lesser part of stomach, 16
peritoneum in, 342	stimulation of, by hormoses, 490
pylorospasm in, 324, 325	by myenteric reflex, 12, 13, 14
source of pain in, 316, 322	Peristaltic ring, 14
von Leube's, 332	Peristaltic waves, action of, in di-
vagotonic influences in, 265	gestion, 2, 14

Peristaltic waves, cont. Polypoid growths, in stomach, inauguration of, 11 398, 399 Peritonitis, abdominal pain in, Polyps, in stomach, 465 656 Popielski, on gastric juice, 20 general, due to perforating ul-Post-operative vomiting, 637 cer, 353 Post-traumatic vomiting, 637 Pernicious anemia, achylia gas-Poultices, for peptic ulcer, 326, trica in, 604, 605, 606 332 in relation to gastric digestion, Pregnancy, and lactation, gastric ulcer, recurring acute, on Petrissage, 551 cessation of, 271 "Phantom tumors," 465 anemia with grave pyloric Pharynx, action of, in digestion, spasm in, 178, 179 post partum hemorrhage, with Phenol, use of, in peptic ulcer, pyloric spasm, in, 178, 179 formula for, 313 relief of vomiting by interrup-Phenolphthalein, use of, in peptic tion of, 641 vomiting in, caused by, 439, 440 ulcer, 325 Phlegmonous gastritis, 559 due to hysterical conditions, Phloroglucinvanillin, 49, 50 641 Phthisis, treatment of post-prannot dependent on, 639 dial vomiting in, 639 pernicious, 640, 641 Physostigmin, action of, on nerve tractable forms of, 642 systems, 106 treatment of, 642 Plexus, Auerbach's, 11 vomiting throughout, with py-Meisner's, 11 loric spasm, 178, 179 myenteric, 11 Pressure, in massage, 552 Plicae-villosae, 7 instruments for measuring de-Pneumococcus infection of stomgree of, 42 ach, causing gastritis, 561 peptic ulcer under, 42 Point, McBurney's, 662 sensitiveness to, 41 of Lang, 662 "Pretubercular dyspepsia," 204 Points of tenderness, iliac, 662, Psoriasis, treatment of, by diet, 665 223 Lang's, 662, 665 Psychasthenia, cure of, by psycho-McBurney's, 662, 665 therapy, 130 Psychosis, with paresthesia gasmesenteric, 662, 665 trica, 190 Poisoning, food, 108 Polyadenomata, of stomach, 464 Psychotherapy, cure by, of mor-Polycythemia, hematemesis with, bid fear of drinking water, 130 for paresthesia gastrica, 190 Polygrams, movements of stomach Ptosis of stomach, 436 studied by, 88 Ptyalin, amylolytic power of, 15 Polyphagia, 195 treatment of, 196 Pulmonary phthisis, condition of stomach in, 131 Polypi, of stomach, 464 Polypoid gastritis, 577 symptoms of, 132

5.	<b></b>
Pulmonary phthisis, cont.	Pyloric stenosis, cont.
treatment of, 133	importance of proper treatment
Pulmonary tuberculosis, func-	of, 367
tional derangement in, 205	of infancy, 628
gastric contents in, 205, 206	anatomical features of, 628
gastric symptoms associated with, 203, 204	characteristic manifestations of, 628
gastric symptoms of, 204, 205, 206	clinical features of, 628 functional features of, 628
psychic state, disturbance of, by	hypertrophy in, 628
gastric symptoms in, 205 psychic state in relation to eat-	theories accounting for, 629
ing in, 206, 207	impermeability of pyloric canal in, 628
stomach in relation to, 203	nature of, 628
treatment of, in relation to gas-	pyloric tumor in, 628
tric symptoms, 206, 207	symptoms of, 630
Purgatives, in peptic ulcer, 318,	treatment of, 630
325, 326	radiograph representing, 365
Pylorie obstruction, due to gastric	resulting from ulcer, 291, 292
ulcer, 360	signs of advance of, 364
due to peptic ulcer, 235, 236	treatment of, 365
indications of, 48, 49	before operation, 366
symptoms of, 363	by dilatation, 370
Pyloric spasm, accompanied by	dietary, 368, 369
over-secretion, 178	general, 369
associated with dyspepsia, 126 associated with hyperchlorhy-	successful methods of, 367, 368, 369
dria, 177	surgical, 370
case of, cited, 178, 179	tubercular, 209
degrees of, 177	Pyloric ulcer, contingent with py-
description of, 176, 177	loric stenosis, 412
followed by gastric stasis, 177	"Pyloric vein," 11, 298
in cholecystitis, 114, 115	Pyloroptosis, 6
indicated by X-ray shadows,	Pylorospasm, 179
<b>94</b> .	indications of, 49
nature of, 176	prognosis of, 181, 182
operative cure of, 126	symptoms and signs of, 181
source of, 177, 178	treatment of, 182
Pyloric stenosis, contingent forms	in peptic ulcer, 324, 325
of, 412	Pylorus, center of, 5
diet for, 368, 369	closing of, in digestion, 18
differential diagnosis between	digestion in, 17, 18
different forms of, 411, 412	dilatation of, 370
due to cancer, 401	nerve control of, 21
due to gastric ulcer, 360, 361	obstructions of, indicated by X-
hyperplasia of muscular wall in,	ray shadows, 94
628	palpation of, 37, 38

Pylorus, etc., cont. position of, 4, 5, 6 spasm of, 176 Pyorrhea alveolaris, organisms in pus of, 373, 374 with gastric erosions, 373 R

Radiograms, importance of interpretation of, 87 interpretation of, 90 Radiographic examination, diagnostic value of, 84, 87 Radiographic shadows, adhesions from pericholecystitis indicated by, 94 alimentary canal, representation of position and movement of, by, 83 atonic stomach indicated by, 95 by bismuth, 83, 84 by gas distension, 83 cancer of stomach indicated by, 100 differentiation of, 89 double capsule means of recognizing slow motion in, 89 duodenal ulcer indicated by, 98 expedients for producing, 83 gastrectasis indicated by, 96 gastric ulcer indicated by, 96 in duodenum, 93 irritable stomach indicated by, 95 modifications of, in shape of stomach, 84, 87, 90, 93 over-tonic stomach indicated by. pyloric obstruction indicated by, pyloric spasm indicated by, 94 Radiography, 83 advantages of, 87 in cancer, 414 preference over fluoroscopy, 87 Rectal alimentation, 727

Rectal alimentation, cont. in peptic ulcer, 331, 332, 340, Recurring vomiting, 643 in Lysteria, 644 Regurgitation, 175 in diagnosis, 28 Reichmann, on gastrosuccorrhea continua, 481 Reichmann's disease, see Gastrosuccorrhea. Relaxation of stomach, 502 Remastication, 175 "Remnant test." 65 Remond and Mathieu, "remnant test" of, 65 Renal colic, differentiated from peptic ulcer, 309 Renal disease, effect on, of condition of stomach, 214 stomach in relation to, 212 Renal insufficiency, 212, 213. 214Rennet zymogen, 59 Rennin and its zymogen, 58 quantitative estimation of Boas' method, 59 Resorcin solution, test to determine free hydrochloric acid. Respiratory diseases, due to indiscreet eating, 199 effect of eating on, 199, 200 stomach in relation to, 199 "Rest," afforded by gastro-enterostomy, 257 for peptic ulcer, 318, 319 details of, 320, 321 importance of complete, 318, 319, 329 Lenhartz on, 334 Von Leube on, 333 "Rest cure," for gastric neurosis, 130 for Graves' disease, 134

Rest in bed, for dyspepsia and

lumbar backache, 127

Revidtzev, on deglutition sounds, Sarcoma of stomach, 460 clinical history of, 462 "Rheumatoid arthritis," 219 diagnosis of, 462 Riegel, on alkalinity of blood metastases with, 461, 462 mixed types of, 461 theory, 248 on gastric ulcer, 279, 280 morbid anatomy of, 460 gastrosuccorrhea continua, round cell type of, 460 spindle cell, 461 test meal of, 48 symptoms of, 462 Rocahout, 724 treatment of, 463 Rochelle salts, for constipation in Schreiber's sound, in diagnosing cardiospasmus, 672 peptic ulcer, 318, 325 Rosenheim, on hyperchlorhydria Scirrhous carcinoma, 403 as cause of peptic ulcer, Sclerosis of abdominal vessels, dif-256 ficulty of clinical diagnosis Rossbach, on gastrosuccorrhea peof, 616 involvement of large branches, riodica, 484 of, 615 Rumination, 175 Rupture of esophagus, by vomitsymptomatology of, 615 ing, 694, 695 Sclerotic gastritis, 559 Secondary gastritis, 559 cases cited of, 696 prognosis of, 697 Secretin, 134 Secretions, 16 spontaneous, 693 symptomatology of, 696 anomalies of, 156 traumatic, 693 disorders of, 102 treatment of, 697 intermittent application of Russian oil, use of, in peptic ulterm, 102 internal, as related to dyspepcer, 327 sia, 133, 134 diseases connected with dis-S turbances of, 133, 134 irregular, indication of treat-Sahli method, for quantitative esment of peptic ulcer. timation of hydrochloric 312 acid, 51 minor importance of disturb-Saline injections, use of, in hemorances in, 72 rhage, 329 Sensation, disorders of, 102 Saline purgatives, use of, in pepneuroses affecting, 185 tic ulcer, 325, 350 Sensitiveness to pressure, under Salivary glands, secretion of, in palpation and percussion. digestion, 1 41 Salomon's test, for cancer, 445, Serous coat of stomach, involve-446 ment of, in peptic ulcer, for carcinoma, 61 treatment for, 341 Sapremia, vomiting due to, 640 Serum, control of hemorrhage by. Saprophytic bacteria, 467 Sarcinae, 63 use of, in peptic ulcer. 330

"Serum disease," 110 Sex, consideration of, in peptic ulcer, 228 Shaking, in massage, 552, 553 Sialorrhea, 522 "Sieving pail," Bassler's, for lavage water, 376 Sight, digestion affected by disturbances of, 140 Silver nitrate, use of, in peptic ulcer, 313, 324 Sinusitis, gastric distress accompanying, 139 Sippy, method of, for dilatation of esophageal stricture, 686, 687, 688, 689 on "string test," 79 Skin diseases, difficulties in treatment of, 220, 221 in relation to digestion, 220 Sleep, in diagnosis, 29, 30 induction of, 194 Smell, dyspepsia arising from disturbance of, 139 sweat reflex arising from, 139 Sodium bicarbonate, use of, in peptic ulcer, 317 Solutions: Günzburg's, 49 Lugol's, 59 resorcin, 50 Soper, on epigastric hernia as cause of peptic ulcer, 261 Sound, danger in use of, for diagnosing cardiospasmus, 672 use of, in diagnosing cardiospasmus, 672 Spasm, indicated by radiograms, indicated by stomach tube, 67, 68, 69 of cardia, 179, 180 of pylorus, 176 treatment of, in peptic ulcer, 324, 325 Special senses, disturbances of, in relation to dyspepsia, 139

Splanchnics, motor inhibition conveyed by, 15 Splanchnoptosis, of Stiller, 530 Spleen, position relative to fundus. 4 under palpation, 40 Squamous-celled carcinoma, 403 Starch digestion, test for, by Lugol's solution, 59 Starling, on gastric juice, 19 Stasis, effect of, on peptic ulcer, 256, 257 Statistics, cancer, distribution of, urban or rural, 382, 383 localization of, as to organs affected, 383, 384, 385 mortality from, 381, 382 Stenosing gastritis, 574 contingent with pyloric stenosis, due to gastric ulcer. 361 Stenosis, indicated by radiograms, Stereoscope, use of, 88, 89 Sternum, position of, relative to stomach, 5 Stethoscope, use of, in auscultation, 45 Stethoscopy, associated with friction, 46 Stiller, splanchnoptosis of, 530 Stomach, actinomycosis of, 468 adenoma in, 398, 399 anatomical description of, 539, 540 anatomy of, 1 areolar or submucous coat of, 7 at rest, 18 atonic, indicated by X-ray shadows, 95 auscultation over, 45 axes of, in considering shape, 6 benign tumor of, 463 bilocular, 290, 291, 359 blood supply of, 10 cancer of, 378 cells of, 7, 8

Stomach, etc., cont. Stomach, etc., cont. condition of, in febrile condimovements of, represented by tions, 217 fluoroscopy, 88 cul-de-sac of, 4 represented by means of polycurvatures of, 5 grams, 88 dilatation of, in relation to gasmucosa of, 7 tric atony, 492 muscular layer of. 7 downward descent of, 538 mycelium in, 468 effect of alcohol on, 578 mycosis of, 467 nerve supply of, 11 elastic tissue maintaining tonicity of, 488, 489 outline of, determined by peremptying of, in digestion, 18 cussion, 43 endomyces albicans of, 467 over-tonic, indicated by X-ray favus of, 468 shadows, 95 fibers of, 6, 7 physiology of, 1 "fish-hook" type of, 358, 538 polypoid forms in, 398, 399 foreign bodies in, 466 position of, 3, 4, 5, 6, 537 fundus of, 539 protecting anti-body in, 267 gas in, 18, 19 ptosis of, 436 glands of, 7, 8 rare form of inflammation of, hourglass, type of, 290, 291, 358 in diabetes, 214 resistance of, to trauma, 249, in malaria, 223 250 in relation to arthritis, 218 saprophytic bacteria in, 467 in relation to cardiac disease. sarcoma of, 460 210 secretions of, 8 in relation to other diseases, shape of, 3, 4, 5, 6 199 shape of during active digesin relation to pulmonary tubertion, 19 culosis, 203 slow motion of, double capsule relation to renal disease, in means of recognizing, 89 sympatheticotonic. states of: 104, 105 in relation to respiratory diseases, 199 vagotonic, 104, 105 "steer-horn" type of, 537 in tuberculosis, 132 involvement of muscle coats of, structure of, 6 syphilis of, 620 tonicity of, 12 irritable, indicated by X-ray shadows, 95 topography of, 538, 539 lower border of, in palpation, tuberculosis of, 208 tuberculous lesions of, 132 36 tubes of, 7 lymphatics of, 10 medium line of, 4 tumors of, other than carcinomatous, benign, 463 modifications in shape of, 84, 87, 90, 93 sarcoma, 460 momentary inhibition of movetwo parts of, 15, 16 ments of. 3 types of, 87, 90

Stomach, etc., cont. umbilicus as fixed point in topography of, 538 varices of, 575 vascularity of, 10 vertical, 358 types of, 537 Stomach contents, in acute catarrhal gastritis, 564 bacterial flora in, 450 indications from, by stomach tube, 68, 69, 70, 71, 72 Stomach symptoms, see Gastric symptoms. Stomach tube, conclusions from, not final in single examination, 69 counter-indications for, 75 dangers of, in diagnosis, 73 diagnostic value of, 72, 73 former reluctance to use of, in open ulcer, 278 indications by introduction of, 67, 68, 69, 70, 71, 72 introduction of, 67, 73 resentment of stomach towards. 564 specifications for, 67 stomach contents found by, 68, technique of use of, 73 use of, 67 in aerophagy, 523 in gastrectasis, 510 in gastrosuccorrhea, 349 in obstruction of esophagus, in study of gastric ulcer, 278 Stone in bladder, associated with dyspepsia, 129 cure of, by lithotrity, 129 Stools, in diagnosis, 29 Stricture, indicated by stomach tube, 68 String test, applications of principle of, 79 description of, 77

String test, etc., cont. incidence of duodenal ulcer by means of, 301, 303 peptic ulcer indicated by, 78 use of, 77 Stupes, use of, see Poultices. Styptin, use of, in peptic ulcer, 328 Superalimentation, 710, 711 caloric values in, 711 for tuberculosis, caution in use of, 204 Surface friction, 551 Surgery, in cancer, importance of early diagnosis for, 410, 411 in peptic ulcer, 329, 330, 334 chronic, 345, 346, 347 in perforating ulcer, 353 in pyloric stenosis, 370 mistaken use of, in gastric disorders, 123, 124, 125, 126 Sweat reflex, 139 Sympathetic gastritis, 559 Sympathetic system of nerves, action of, 104 Sympathetic vomiting, 636 Sympatheticotonic state, in dyspepsia, 155 of stomach, 104, 105, 106 Syphilis, mistaken for malignancy, 623, 624, 625 resemblance of, to cancer, 440 Syphilis of stomach, see Gastric syphilis. Syphilitic ulceration, 620

## T

Tachycardia, due to gastric disturbance, 210, 211
paroxysmal, treatment of, 212
Tannic acid, use of, in peptic ulcer, 332
Taste, disturbances of, 140
Tea, 725
Teeth, faulty, disturbance of digestion by, 1

Temperature, high, diminishing of Tissues, resistance of, to gastric hydrochlorie acid in, 217 juice, 247, 248, 266, 267 Tenderness, abdominal, 649 Toast, 722 elicited by deep pressure, 665 Tonicity, sources of, 13, 14 location of seat of, 662 Tonus, 12 with angor abdominalis, 655 of gastric walls, 12, 13, 14 with gastric neuroses, 665 transient relaxation of, 22 points of, 662, 665 Töpfer's method for quantitative various conditions of, 665 estimation of hydrochloric Test meals: Ewald, 48 acid, 51 Germain Seé, 48 Torpid liver, 111, 112 Torsion of ureter, differentiated Riegel, 48 Benzidin, for occult blood, 61 from perforating ulcer, 355 Tests: Biuret, for peptones, 55 Total acidity, quantitative estimation of, Töpfer's method. Boas', for motor activity of stomach, 65 51, 52, 53 cancer, 445, 446, 447 variation in, 279, 280 dimethylamidoazobenzole, 50 Toxemia, in cancer, treatment of, for motor activity of stomach, 456 Toxic gastritis, 559, 568 Guiaiacum, for occult blood, Trauma, in peptic ulcer, 249 resistance of stomach to, 249, Günzburg's solution, for free 250 hydrochloric acid, 49, 50 "Trophic neurosis," 268 Kelling's, for determining lactic Trousseau's bougie, in diagnosing acid, 54 cardiospasmus, 672 qualitative, for free HCl, 49, Tryptophan test, for carcinoma, 50 for pepsin, 55 Tube, stomach, see Stomach tube. for pepsinogen, 55 Tubercular pyloric stenosis, cause "remnant," 65 of, 209 resorcin solution, for free hyprognosis of, 210 symptoms of, 209, 210 drochloric acid, 50 Salomon's, for carcinoma, 61 Tuberculosis, dyspepsia associated with, 131, 132, 133 string, 77 tryptophan, for carcinoma, 61 importance of nourishment in. Uffelmann's, for determining 131 of stomach, see Gastric tuberlactic acid, 53 Therapeutics, principle of, Debout culosis. d'Estrées, 138 pulmonary, stomach in relation Thoracic disease, abdominal pain to, 203 Tuberculous lesions of stomach, in, 657 Thorax, position of, in relation to 132 fundus, 4 Tumors, benign, of stomach, see Benign tumors of stomach. Thrush, 561 Tissier, Paul, on acute gastrecfalse, 465 tasis, 516 inflammatory, deep-seated, 413

Tumors, etc., cont.
resembling cancer, case cited,
413
phantom, 465
Tumors of stomach, difficulties in
diagnosis of, 466
other than carcinomatous, benign, 463
sarcoma, 460
Turck, F. B., on bacterial infection in peptic ulcer, 253
Typhoid ulcer, 296
in esophagus, 682

### U

Uffelman's test, for determining lactic acid, 53 Ulcer, atropin for, 266 cancer from scar of, 415 cancer growing upon, 414, 416, cancer preceding, 415 classed as mere vagotonic manifestation, 265, 266 duodenal, 294 See Duodenal ulcer. gastric, see Gastric ulcers. indicated by stomach tube, 71 nervous system in relation to, 262 onset of recent, 273 recurring, 286 Ulceration, causes of, 228 peptic ulcer differentiated from other forms of, 228 process of, 228 Ulcus carcinomatosum, 414, 416, Umbilicus, as fixed point in topography of stomach, 538 position of, 5, 6 Uremia, gastritis from, 578 gastrititis with, 561 in duodenal ulcer, 296 vomiting in, 213 Uremic vomiting, 634

Uremic vomiting, etc., cont.
peculiarities of, 635
Ureter, torsion of, differentiated
from perforating ulcer, 355
Uricacidemia, associated with dyspepsia, 129
cure of, by lithotrity, 129
Urinary tract, dyspepsia due to irritations of, 128
Urticaria, cause of, 222

# V

Vagotonic states, in dyspepsia, in hyperchlorhydria, 159 of stomach, 104, 105, 106 Vagus, action of branches of, 104 effect of eating on, 2 functions of, 2, 13, 14, 15 innervation from, 2 stimulating gastric juice, 20 tonicity affected by, 13, 14 Varices of stomach, 575 Vascular obstruction, as causing peptic ulcer, 260 Vegetables, 718 mastication of, 1 Vertebrae, position of, in relation to stomach, 4, 5 Vibratory pressure, in massage, 552 Vibration, 552, 553 Vilbration, 194 Virchow, connective tissue theory of, on cancer, 378 Visceral innervation, 106 Visceroptosis, 536 Vomiting, 632 associated efforts of, 633 cerebral, 433 cyclic, 643, 646 of childhood, 644, 645 treatment of, 647 dependent on auto-suggestion, due to chlorosis, 634

Vomiting, cont. due to eye-strain, 636 due to gastric irritation, 634 due to metabolic causes, 636 due to sapremia, 640 due to spinal irritation, 634 explanation of, 433 fecal, 636 general principles, in treatment of, 648 in cancer of stomach, 432 in cholecystitis, 635 in diagnosis, 27, 28 in dilatation of stomach, 506 in gastric ulcer, 284, 285, 286 significance of, 286 in gastrosuccorrhea, 349 in infants with duodenal ulcer, 294 in malaria, 224 in peptic ulcer, 312, 327 in perforating ulcer, 352 in pregnancy, 439, 440 due to hysterical conditions, 641 not depending on it, 639 pernicious, 640, 641 tractable forms of, 642 treatment of, 642 mal de mer, 638 mechanism of, 633 obstinate cases of, 640 obstructive, 635 of ileus, 635 organism implicated in, 632 periodic, 648 post-operative, 637

Vomiting, cont. post-prandial vomiting of phthisis, 639 post-traumatic, 637 recurring, 643 reflex, 635 sympathetic, 636 throughout pregnancy, with pyloric spasm, 178, 179 treatment of, 637, 638 in cancer, 455 in peptic ulcer, 327 uremic, 634 Vomitus, appearance of, 476 blood in, 476 Von Leube treatment, for gastric ulcer, 332

# W

Water, 726
drinking of, in diabetes, 215
morbid fear of drinking, 130
Williams, H. U., on cancer, pathology of, 397
photomicrographs of gastric erosions by, 375

Y

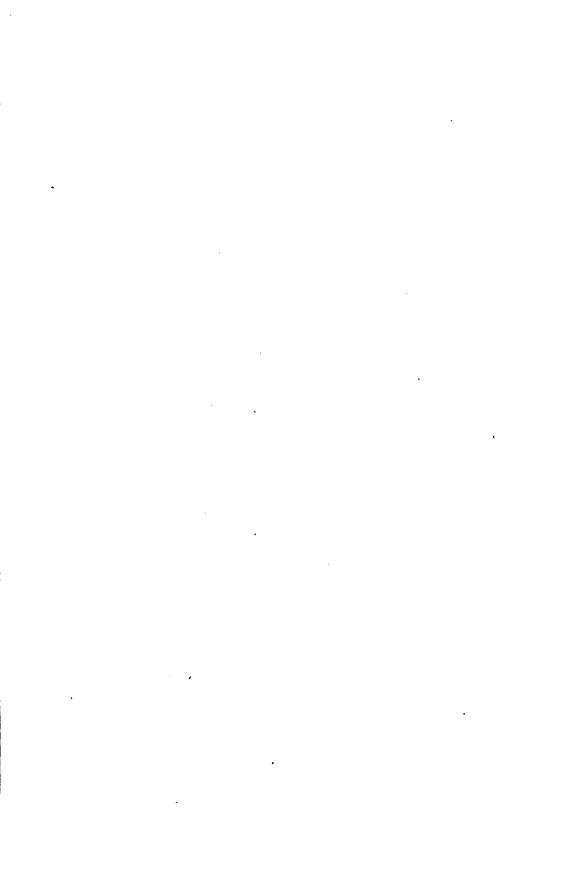
Yeast cells, 63

7

Zweiback, 721, 722 Zymogen, of rennin, 58, 59 Zymogen pepsinogen, 54, 55 Zymogens, 54

. • 

		-
	•	



Proping Maspiral

--



# Lin. Couldins' v



